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1864.

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1808. MATTHEW BAILLIE, M.D.
1810. SIR HENRY HALFORD, Bart., M.D., G.C.H.
1813. SIR GILBERT BLANE, Bart., M.D.
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1817. WILLIAM BABINGTON, M.D.
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1821. JOHN COOKE, M.D.
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1841. ROBERT WILLIAMS, M.D.
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OCTOBER 1864.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents, those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1841 *James Abercrombie, M.D., Cape of Good Hope.

1846 *John Abercrombie, M.D., Physician to the Cheltenham General Hospital, 13, Suffolk square, Cheltenham.

1851 *Henry Wentworth Acland, M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Physician to the Radcliffe Infirmary; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.

1847 Elisha Acosta, M.D., New York, U.S.

1842 William Acton, 17, Queen Anne street, Cavendish square. *Trans.* 1.

Elected

1851 JOHN ADAMS, Surgeon to, and Lecturer on Descriptive and Surgical Anatomy at, the London Hospital; 4, St. Helen's place, Bishopsgate street. *Trans.* 3.

1852 WILLIAM ADAMS, Surgeon to the Royal Orthopædic Hospital; 5, Henrietta street, Cavendish square. *Trans.* 2.

1837 *RALPH FAWSETT AINSWORTH, M.D., Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1839 SIR RUTHERFORD ALCOCK, K.C.B., K.C.T., K.T.S., H.M.'s Envoy Extraordinary and Consul-General in Japan. *Trans.* 1.

1826 JAMES ALDERSON, M.D., F.R.S., Senior Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; 17, Berkeley square. S. 1829. C. 1848. T. 1849. V.P. 1852-3. *Trans.* 3.

1843 CHARLES JAMES BERRIDGE ALDIS, M.D., Medical Officer of Health for St. George's, Hanover square; Senior Physician to the Surrey Dispensary; and Physician to the St. Paul and St. Barnabas Dispensary; 1, Chester terrace, Chester square. *Trans.* 2.

1850 CHARLES REVANS ALEXANDER, Surgeon to the Royal Infirmary for Diseases of the Eye; 6, Cork street, Bond street.

1863 JULIUS ALTHAUS, M.D., 18, Bryanston street, Portman square.

1862 EDWYN ANDREW, M.D., Windsor House, Shrewsbury.

1862 JAMES ANDREW, M.D., Assistant Physician to, and Warden of the College, St. Bartholomew's Hospital.

1820 THOMAS ANDREWS, M.D., Norfolk, Virginia.

1819 †JAMES MONCRIEFF ARNOTT, F.R.S., Surgeon Extraordinary to H.M. the Queen; 2, New Burlington street. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Trans.* 8.

1817 †JOHN ASHBURNER, M.D., F.L.S., 7, Hyde park place, Cumberland gate. C. 1821, 1830-1.

1851 THOMAS JOHN ASHTON, 31, Cavendish square.

Elected

1825 †Benjamin Guy Babington, M.D., F.R.S., Physician to the Asylum for Deaf and Dumb; Consulting Physician to the German Hospital, and to the City of London Hospital for Diseases of the Chest; 31, George street, Hanover square. C. 1829. V.P. 1845-6. T. 1848. P. 1861-2. *Trans.* 2

1820 *John Badley, Dudley, Worcestershire.

1840 William Bainbridge, 47, Bridge street, Southwark.

1836 Andrew Wood Baird, M.D., Physician to the Dover Hospital; Dover, Kent.

1851 *Alfred Baker, Surgeon to the Birmingham General Hospital, and Lecturer on Surgery at Sydenham College; Cannon street, Birmingham.

1839 †Thomas Graham Balfour, M.D., F.R.S., Deputy Inspector-General of Hospitals; 10, Sumner place, Onslow square, Brompton. C. 1852-3. V.P. 1860-1. *Trans.* 2.

1848 Edward Ballard, M.D., Medical Officer of Health for Islington; 7, Compton terrace, Upper street, Islington. *Trans.* 2.

1849 Thomas Ballard, M.D., 10, Southwick place, Hyde park.

1847 Andrew Whyte Barclay, M.D., Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Medical Officer of Health for Chelsea; 23A, Bruton street, Berkeley square. S. 1857-60. L. 1861-2. *Trans.* 2.

1848 Edgar Barker, 9, Oxford square, Hyde park.

1862 Edgar Barker, jun., late Surgeon to the Western General Dispensary; 5, Albion place, Hyde park square.

1833 †Thomas Alfred Barker, M.D., Senior Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2 *Trans.* 6.

1843 Thomas Herbert Barker, M.D., F.R.S.E., Senior Surgeon to the Bedford General Dispensary; Harpur-place, Bedford.

Elected

1847 GEORGE HILARO BARLOW, M.D., Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; Physician to the Magdalen Hospital; 12, Union street, Southwark. C. 1859.

1861 ROBERT BARNES, M.D., Obstetric Physician to the London Hospital; Obstetric Physician to, and Lecturer on Midwifery at, St. Thomas's Hospital, and Physician to the Royal Maternity Charity; 46, Finsbury sq. *Trans.* 3.

1864 JOSEPH GILLMAN BARRATT, M.D., 22, Cleveland Gardens.

1840 BENJAMIN BARROW, Surgeon to the Royal Isle of Wight Infirmary; Clifton House, Ryde, Isle of Wight.

1859 RICHARD BARWELL, Assistant-Surgeon to, and Lecturer on Comparative Anatomy at, the Charing Cross Hospital; 32, George street, Hanover square. *Trans.* 1.

1844 WILLIAM RICHARD BASHAM, M.D., *Vice-President*, Senior Physician to, and Lecturer on Medicine at, the Westminster Hospital; 17, Chester street, Grosvenor place. S. 1852-4. C. 1860-1. V.P. 1864. *Trans.* 2.

1862 PIERRE VICTOR BAZIRE, M.D., 28, Woburn square.

1862 LIONEL SMITH BEALE, M.B., F.R.S., Professor of Physiology and General and Morbid Anatomy in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street.

1860 ADAM BEALEY, M.D., M.A. Camb., Physician to the Royal General Dispensary, St. Pancras; 27, Tavistock square.

1841 GEORGE BEAMAN, M.D., 3, Henrietta street, Covent Garden.

1856 AMOS BEARDSLEY, Bay villa grange, Newton in Cartmel, Lancashire.

1836 WILLIAM R. BEAUMONT, Consulting Surgeon to the Toronto General Hospital, late Professor of Surgery in the University of King's College; Toronto, Canada West. *Trans.* 3.

1840 CHARLES BEEVOR, 41, Upper Harley street.

1858 WILLIAM CHAPMAN BEGLEY, M.D., Middlesex County Lunatic Asylum, Hanwell.

1819 †THOMAS BELL, F.R.S., F.L.S., the Wakes, Selborne, Hants. C. 1832-3. V.P. 1854. *Trans.* 1.

Elected

1847 James Henry Bennet, M.D., The Ferns, Weybridge, and Mentone.

1845 Edward Unwin Berry, 38, Gower street, Bedford square.

1820 Stephen Bertin, Paris.

1815 †Archibald Billing, M.D., F.R.S., Member of the Senate of the University of London; 6, Grosvenor gate. C. 1825. V.P. 1828-9.

1827 William Birch, Barton-under-Needwood, Staffordshire. *Trans.* 2.

1855 Peter Hinckes Bird, F.L.S., 1, Norfolk square, Hyde park.

1856 William Bird, Surgeon to the West London Hospital; 7, George street, Hanover square.

1849 Edmund Lloyd Birkett, M.D., Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square.

1851 George Birkett, M.D., Resident Proprietor, Northumberland House, Green Lanes, Stoke Newington.

1851 John Birkett, F.L.S., *Secretary*, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 59, Green street, Grosvenor square. L. 1856-7. S. 1863-4. *Trans.* 4.

1846 Hugh Birt, British Naval Hospital, Valparaiso, Chili.

1843 Patrick Black, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Queen Anne street, Cavendish square. C. 1856.

1847 George C. Blackman, M.D., Professor of Surgery in the Medical College of Ohio; New York, U.S.

1840 Peyton Blakiston, M.D., F.R.S., St. Leonard's-on-Sea.

1845 Henry Blenkinsop, Senior Surgeon to the Warwick Dispensary; Jury street, Warwick.

1823 Louis Henry Bojanus, M.D., Wilna.

1846 John Ashton Bostock, Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 54, Chester square, Belgravia. C. 1861-2.

1863 Francis Bowen, M.D., 62, Upper Berkeley street, Portman square.

1841 William Bowman, F.R.S., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3 V.P. 1862. *Trans.* 3.

Elected

1862 WILLIAM HENRY BRACE, Surgeon to the Bath United Hospital; 1, Gay street, Bath.

1857 WILLIAM BRINTON, M.D., F.R.S., Physician to, and Lecturer on Physiology at, St. Thomas's Hospital; 24, Brook street, Grosvenor square.

1851 BERNARD EDWARD BRODHURST, Assistant Surgeon to St. George's Hospital, and to the Royal Orthopædic Hospital; 20, Grosvenor street. *Trans.* 2; *Pro.* 1.

1844 CHARLES BROOKE, M.A., F.R.S., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 16, Fitzroy square. C. 1855.

1848 WILLIAM PHILPOT BROOKES, M.D.

1854 *HENRY BROWN, Surgeon to H.M. the Queen, and the Royal Household; Windsor.

1857 *ROBERT BROWN, Surgeon to the Carlisle Dispensary; 4, Devonshire street, Carlisle.

1860 CHARLES EDOUARD BROWN-SÉQUARD, M.D., F.R.S.

1851 ALEXANDER BROWNE, M.D., Twynholm, Kirkcudbright.

1860 THOMAS BRYANT, Assistant-Surgeon to, and Demonstrator of Operative Surgery at, Guy's Hospital; 2, Finsbury square. *Trans.* 4; *Pro.* 1.

1855 WALTER JOHN BRYANT, L.R.C.P. Edinb., 7, Bathurst-street, Hyde park gardens.

1823 B. BARTLET BUCHANAN, M.D.

1864 GEORGE BUCHANAN, M D., Physician to the London Fever Hospital, and Assistant Physician to the Hospital for Sick Children; Medical Officer of Health for St. Giles District; 75, Gower-street.

1839 GEORGE BUDD, M.D., F.R.S., Consulting Physician to the Seamen's Hospital Ship 'Dreadnought'; 20, Dover street, Piccadilly. C. 1846-7. V.P. 1857. *Trans.* 5.

1839 THOMAS HENRY BURGESS, M.D., Portsmouth.

1853 PATRICK BURKE, 12, Upper Montagu street, Montagu square.

1833 †GEORGE BURROWS, M.D., F.R.S,, President of the Medical Council, Consulting Physician to St. Bartholomew's Hospital, Physician to Christ's Hospital; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V.P. 1849-50. *Trans.* 2.

Elected

1820 Samuel Burrows.

1837 George Busk, F.R.S., F.L.S., Examiner in Comparative Anatomy at the University of London; Surgeon to the Seamen's Hospital Ship 'Dreadnought'; 15, Harley street, Cavendish square. C. 1847-8. V.P. 1855. *Trans.* 4.

1818 John Butter, M.D., F.R.S., F.L.S., Physician Extraordinary to the Plymouth Royal Eye Infirmary; Windsor villas, Plymouth.

1851 *William Cadge, Surgeon to the Norfolk and Norwich Hospital; All Saints green, Norwich. *Trans.* 1.

1851 Thomas Callaway, Algiers.

1861 George William Callender, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 1.

1852 *George Canney, M.D., Bishop-Auckland, Darlington, Durham.

1847 John Burford Carlill, M.D., Surgeon-Accoucheur to the Newman street Lying-in Institution; 57, Berners street.

1853 Robert Brudenell Carter, Stroud, Gloucestershire.

1845 Samuel Cartwright, Professor of Dental Surgery at King's College, London; Surgeon-Dentist to King's College Hospital; 32, Old Burlington street. C. 1860-1.

1839 William Cathrow, 42, Weymouth street, Portland place. C. 1860-1.

1845 William Oliver Chalk, Surgeon to the St. Marylebone Eye and Ear Institution; 3, Nottingham terrace, York gate, Regent's park.

1844 Thomas King Chambers, M.D., Hon. Physician to H.R.H. the Prince of Wales; Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Physician to the Lock Hospital; 22B, Brook street, Grosvenor square. *Trans.* 1. C. 1861.

1859 Frank Chance, M.D., 51, Wimpole street, Cavendish square.

1849 Frederick Chapman, Old Friars, Richmond green, Surrey.

Elected

1837 HENRY THOMAS CHAPMAN, 16, Lower Seymour street, Portman square. C. 1858.

1852 GEORGE BORLASE CHILDS, Surgeon-in-Chief, to the City Police Force, and Surgeon to the Metropolitan Free Hospital; 11, Finsbury place South.

1842 WILLIAM DINGLE CHOWNE, M.D., Physician to, and Lecturer on Medicine and Midwifery at, the Charing Cross Hospital; Corresponding Fellow of the Royal Academy of Surgery of Madrid; 8, Connaught place West, Hyde park. C. 1853-4.

1860 ANDREW CLARK, M.D., Assistant Physician to the London Hospital; 23, Montague place, Russell square.

1839 FREDERICK LE GROS CLARK, Surgeon to, and Lecturer on Surgery at St. Thomas's Hospital, Surgeon to the Magdalen Hospital; Consulting Surgeon to the Western General Dispensary, and to the London Female Penitentiary, Pentonville; 14, St. Thomas's street, Southwark, and Lee, Kent. S. 1847-9. V.P., 1855-6. *Trans.* 3.

1862 CHARLES HALL CLARKE, M.D., Stoneyhurst College, near Blackburn, Lancashire.

1848 JOHN CLARKE, M.D., Physician to the British Lying-in Hospital, and Physician to the General Lying-in Hospital; 42, Hertford street, May fair.

1861 *WILLIAM JAMES CLARKE, Surgeon to the Huddersfield Infirmary; John William street, Huddersfield, Yorkshire.

1850 JOSIAH CLARKSON, New Hall street, Birmingham, *Trans.* 1.

1842 OSCAR MOORE PASSEY CLAYTON, 87, Harley street.

1853 JOSEPH THOMAS CLOVER, 3, Cavendish place, Cavendish square.

1857 CHARLES COATES, F.R.C.P. Edinb., Physician to the Bath United General Hospital; 10, Circus, Bath.

1851 EDWARD COCK, Senior Surgeon, to, and Lecturer on Clinical Surgery at, Guy's Hospital; Consulting Surgeon to the Asylum for Deaf and Dumb; 11, St. Thomas's street east, Southwark. C. 1857. *Trans.* 3.

Elected

1850 Daniel Whitaker Cohen, M.D.

1835 *William Colborne, Chippenham, Wiltshire.

1818 Robert Cole, F.L.S., Holybourne, Hampshire.

1855 Frederick Collins, M.D., Medical Officer of Health for Wanstead; Wanstead Lodge, Essex.

1828 John Conolly, M.D., D.C.L., Consulting Physician to the Middlesex County Lunatic Asylum, Hanwell.

1840 *William Robert Cooke, Osborne House, Lower Norwood.

1819 George Cooper, Brentford, Middlesex.

1841 George Lewis Cooper, one of the Surgeons to the National Vaccine Institution, and Teacher of Vaccination to the Medical School of University College; Surgeon to the Bloomsbury Dispensary; 7, Woburn place, Russell square. C. 1860-1.

1843 William White Cooper, Surgeon-Oculist in Ordinary to H.M. the Queen; and Hon. Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9.

1841 Holmes Coote, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 13, Queen Anne street, Cavendish square. S. 1853-4. C. 1864. *Trans.* 2.

1835 George Ford Copeland, Cheltenham.

1822 †James Copland, M.D., F.R.S., Consulting Physician to the Royal Infirmary for Children, and to the Great Northern Hospital, King's Cross; Hon. Fellow of the Royal Academy of Sciences of Sweden, &c.; 5, Old Burlington street. C. 1831. V.P. 1838-9. P. 1853-4.

1860 *Thomas Charles Steuart Corry, M.D., Surgeon to the Belfast General Dispensary; 9, Clarendon place, Belfast.

1839 *Charles Cæsar Corsellis, M.D., F.L.S., Benson, Oxon.

1853 William Gillett Cory, M.D.

1847 Richard Payne Cotton, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; 46, Clarges street, Piccadilly. C. 1863.

Elected

1828 †WILLIAM COULSON, Hon. Consulting Surgeon to St. Mary's Hospital, and to the German Hospital; 1, Chester terrace, Regent's park. C. 1831. L. 1832-7. V.P. 1851-2. *Trans.* 1.

1864 WALTER JOHN COULSON, Surgeon to the Lock Hospital, and Surgical Registrar to St. Mary's Hospital; 29, St. James's place.

1860 †JOHN COUPER, Assistant-Surgeon to, and Lecturer on Physiology at, the London Hospital; 33, Finsbury Circus.

1862 GEORGE COWELL, Surgeon to the St. George's and St. James's Dispensary; 4, St. George's square, Pimlico.

1841 MERVYN ARCHDALL NOTT CRAWFORD, M.D., Wiesbaden. C. 1853-4.

1847 GEORGE CRITCHETT, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 75, Harley street, Cavendish square. *Trans.* 1.

1862 SAMUEL CROMPTON, 17A, Princess street, Manchester.

1837 JOHN FARRAR CROOKES, Harewell, near Faversham, Kent.

1860 RICHARD CROSS, M.D., Queen street, Scarborough.

1849 *WILLIAM EDWARD CROWFOOT, Beccles, Suffolk.

1851 JAMES CAMERON CUMMING, M.D., 1, Cadogan place, Sloane street.

1846 HENRY CURLING, Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.

1837 THOMAS BLIZARD CURLING, F.R.S., Surgeon to the London Hospital; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. *Trans.* 13. *Pro.* 1.

1847 JOHN EDMUND CURREY, M.D., Lismore, County Waterford.

1822 CHRISTOPHER JOHN CUSACK, Chateau d'Eu, France.

1852 THOMAS CUTLER, M.D., Acting Physician at the Spa Waters; Spa, Belgium.

1836 *JAMES STOCK DANIEL, Ramsgate, Kent.

1848 HENRY DAUBENY.

1846 FREDERICK DAVIES, M.D., 19, Upper Gower street, Bedford square.

Elected

1847 JOHN DAVIES, M.D., Physician Extraordinary to the Hertford General Infirmary, and Visiting Physician to the Hadham Palace Lunatic Asylum, Hertford.

1853 ROBERT COKER NASH DAVIES, Rye, Sussex.

1852 WILLIAM DAVIES, M.D., 10, Gay street, Bath.

1852 JOHN HALL DAVIS, M.D., Physician Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Physician to the Royal Maternity Charity; and Consulting Physician-Accoucheur to the St. Pancras Infirmary; 11, Harley street, Cavendish square.

1818 JAMES DAWSON, Wray Castle, Windermere.

1847 GEORGE EDWARD DAY, M.D., F.R.S., Emeritus Professor of Medicine in the University of St. Andrew's; St. Mary Church, Torquay.

1858 TEOFILO DELIMA, M.D., Caracas, Venezuela, South America.

1846 *SAMUEL BEST DENTON, M.D., Ivy Lodge, Hornsea, Hull.

1859 WILLIAM HOWSHIP DICKINSON, M.D., Curator of the Pathological Museum at St. George's Hospital; Assistant-Physician to the Hospital for Sick Children; 11, Chesterfield street, May fair. *Trans.* 5.

1844 ROBERT DICKSON, M.D., F.L.S., Physician to the Scottish Hospital; 16, Hertford street, May fair. C. 1860.

1839 †JAMES DIXON, *Treasurer*, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Consulting Ophthalmic Surgeon to the Asylum for Idiots; 2, Portman square. L. 1849-55. V.P. 1857-8. T. 1863-4. *Trans.* 4.

1862 HORACE B. DOBELL., M.D., Physician to the Royal Infirmary for Diseases of the Chest, City road; 41, Harley street. *Trans.* 1.

1845 JOHN DODD.

1857 ARCHIBALD DOUGLAS, M.D., 8, Clifton place, Sussex square, Hyde park.

1863 JOHN LANGDON HAYDON DOWN, M.D., Assistant-Physician to, and Lecturer on Materia Medica and Therapeutics at, the London Hospital; Physician to the Asylum for Idiots, Earlswood, Redhill. *Trans.* 1.

Elected

1853 Robert Druitt, M.R.C.P., Medical Officer of Health for St. George's, Hanover square; 37, Hertford street, May fair. *Trans.* 2.

1845 George Duff, M.D., High Street, Elgin.

1845 Edward Willson Duffin, 18, Devonshire street, Portland place. *Trans.* 1.

1833 Robert Dunn, 31, Norfolk street, Strand. C. 1845-6. *Trans.* 2.

1861 Claudius Francis Du Pasquier, Surgeon-Apothecary to H.M. the Queen; and to the Household of H.R.H. the Prince of Wales; 62, Pall Mall.

1863 Arthur Edward Durham, Assistant Surgeon to, and Lecturer on Anatomy at, Guy's Hospital, 30, Brook street, Grosvenor square. *Trans.* 2.

1843 Christopher Mercer Durrant, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1839 Henry Sumner Dyer, M.D., 37, Bryanston sq. C. 1854-5.

1836 James William Earle, late of Norwich.

1824 George Edwards.

1823 Charles Chandler Egerton, Kendall Lodge, Epping.

1861 *Robert Elliot, M.D., Physician to the Carlisle Dispensary; 18, Lowther street, Carlisle.

1848 George Viner Ellis, Examiner in Anatomy at the University of London; Professor of Anatomy in University College, London; University College, Gower street. C. 1863-4. *Trans.* 2.

1854 *James Ellison, M.D., Surgeon in Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1835 William England, M.D., Ipswich, Suffolk.

1842 John Erichsen, Professor of Surgery in University College, London, and Surgeon to University College Hospital; Examiner in Surgery to the Royal College of Physicians; 6, Cavendish place, Cavendish square. C. 1855-6. *Trans.* 2.

1836 George Fabian Evans, M.D., Physician to the General Hospital, Birmingham.

1815 *Griffith Francis Dorsett Evans, M.D. C. 1838.

1845 William Julian Evans, M.D., Pinner, Middlesex.

Elected

1864 CHARLES HILTON FAGGE, M.D., Demonstrator of Anatomy at Guy's Hospital; Physician to the Royal Infirmary for Diseases of Children and Women, Waterloo road; 43, Trinity square, Southwark.

1858 RANDLE WILBRAHAM FALCONER, M.D., Physician to the Bath United Hospital; 22, Bennett street, Bath.

1862 ROBERT FARQUHARSON, M.D., Coldstream Guards' Hospital, Vincent square, Westminster.

1844 ARTHUR FARRE, M.D., F.R.S., *Vice-President*, Physician-Accoucheur to H.R.H. the Princess of Wales and H.R.H. the Princess Louis of Hesse; 12, Hertford street, May fair. C. 1857. V.P. 1864.

1863 SAMUEL FENWICK, M.D., 74, Harley street, Cavendish square. *Trans.* 1.

1831 †ROBERT FERGUSON, M.D., Physician Extraordinary to H.M. the Queen, and Consulting Physician to King's College Hospital; 14, Curzon street, May fair. C. 1839. V.P. 1847.

1841 WILLIAM FERGUSSON, F.R.S., *Vice-President*, Surgeon Extraordinary to H.M. the Queen; Professor of Surgery in King's College, London, and Surgeon to King's College Hospital; Professor of Anatomy and Surgery at the Royal College of Surgeons; 16, George street, Hanover square. C. 1849-50. V.P. 1863-4. *Trans.* 4.

1852 *ALFRED GEORGE FIELD, Surgeon to St. Mary's Hospital, Brighton; 28, Old Steine, Brighton.

1849 GEORGE TUPMAN FINCHAM, M.D., Physician to, and Lecturer on Medical Jurisprudence at, the Westminster Hospital; 2, Eccleston terrace south, Eccleston square.

1836 SIR JOHN WILLIAM FISHER, Surgeon-in-Chief to the Metropolitan Police Force; 5, Grosvenor gate. C. 1843-4.

1860 THOMAS GEORGE FITZGERALD, Staff-Surgeon; 6, Whitehall yard.

1842 THOMAS BELL ELCOCK FLETCHER, M.D., Physician to the Birmingham General Hospital; Waterloo street, Birmingham. *Trans.* 1.

1859 WILLIAM HENRY FLOWER, F.R.S., Conservator of the Museum at the Royal College of Surgeons of England. *Trans.* 1.

Elected

1848 JOHN GREGORY FORBES, Surgeon to the Metropolitan Convalescent Institution; 9, Devonport street, Hyde park. *Trans.* 2.

1852 †JOHN COOPER FORSTER, Assistant Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Royal Infirmary for Children, &c.; 10, St. Thomas's street, Southwark. *Pro.* 1.

1859 EDWARD LONG FOX, M.B., Physician to the Bristol Royal Infirmary; 1, Chesterfield place, Clifton.

1858 *WILSON FOX, M.D., Professor of Pathological Anatomy at University College, London, and Assistant-Physician to University College Hospital; 24, Henrietta street, Cavendish square. *Trans.* 2.

1841 JOHN CHRISTOPHER AUGUSTUS FRANZ, M.D.

1843 PATRICK FRASER, M.D., Physician to the London Hospital, and to the London Dispensary; 63, Grosvenor street.

1836 †JOHN GEORGE FRENCH, Surgeon to the St. James's Infirmary: 41, Great Marlborough street. C. 1852-3.

1849 ROBERT TEMPLE FRERE, M.A., F.R.C.P., 34, Upper Harley street.

1846 HENRY WILLIAM FULLER, M.D., *Secretary*, Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; 13, Manchester square. C. 1862. S. 1864. *Trans.* 2.

815 *GEORGE FREDERICK FURNIVALL, Medical Attendant of Great Foster House Asylum for Lunatics; Egham, Surrey.

1864 *WILLIAM TENNANT GAIRDNER, M.D., Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary; 21, Blythswood square, Glasgow.

1860 ROBERT CAMERON GALTON, M.D., Hadzor House, Droitwich.

1854 ALFRED BARING GARROD, M.D., F.R.S., Examiner in Materia Medica at the University of London; Professor of Materia Medica in King's College, London, and Physician to King's College Hospital; 84, Harley street, Cavendish square. *Trans.* 8.

Elected

1857 GEORGE GREEN GASCOYEN, Surgeon to the Lock Hospital; Assistant-Surgeon to, and Lecturer on Descriptive and Surgical Anatomy at, St. Mary's Hospital; 48, Queen Anne street, Cavendish square. *Trans.* 1.

1851 GEORGE GASKOIN, 3, Westbourne park.

1819 HENRY GAULTER.

1848 JOHN GAY, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 10, Finsbury place south.

1821 *RICHARD FRANCIS GEORGE, late Senior Surgeon to the Bath General Hospital; 20, Marlborough Buildings, Bath.

1864 GEORGE DUNCAN GIBB, M.D., M.A., Assistant-Physician to, and Lecturer on Forensic Medicine at, the Westminster Hospital; 19A, Portman street, Portman square.

1858 BENJAMIN GODFREY, M.D., Carlton House, Enfield, Middlesex.

1851 STEPHEN JENNINGS GOODFELLOW, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 5, Savile row, Burlington Gardens. C. 1864. *Trans.* 2.

1818 JAMES ALEXANDER GORDON, M.D., C.B., F.R.S., Pixholme, near Dorking, Surrey. C. 1828. V.P. 1829. *Trans.* 1.

1862 JOHN G. GOULSTONE, M.D., Bellevue, Shrewsbury.

1851 PETER YEAMES GOWLAND, Surgeon to St. Mark's Hospital; 34, Finsbury square.

1844 JOHN GRANTHAM, Crayford, Kent.

1846 GEORGE THOMPSON GREAM, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; 2, Upper Brook street, Grosvenor square. C. 1863.

1843 ROBERT GREENHALGH, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital, and Physician to the Samaritan Free Hospital for Women and Children; 76, Grosvenor street.

1860 EDWARD HEADLAM GREENHOW, M.D., Assistant-Physician to, and Lecturer on Public Health and on Medical Jurisprudence at, the Middlesex Hospital, and Consulting Physician to the Western General Dispensary; 77, Upper Berkeley street, Portman square. *Trans.* 2.

Elected

1814 JOHN GROVE, M.D., Salisbury.

1852 JOHN GROVE, West Hill, Wandsworth, Surrey.

1860 HENRY GUENEAU DE MUSSY, M.D., 4, Cavendish place, Cavendish square.

1849 WILLIAM WITHEY GULL, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 26, Brook street, Grosvenor square. C. 1864. *Trans.* 2.

1837 JAMES MANBY GULLY, M.D.; Great Malvern, Worcestershire.

1859 THEOPHILUS MILLER GUNN; 40, York place, Portman square.

1854 SAMUEL OSBORNE HABERSHON, M.D, Assistant-Physician to, and Lecturer on Materia Medica and Therapeutics at, Guy's Hospital; 22, Wimpole street, Cavendish square. *Trans.* 2.

1849 HAMMETT HAILEY, Newport Pagnell, Bucks.

1845 SIR JOHN HALL, M.D, K.C.B., Inspector-General of Hospitals; 36, Necker Strasse, Stuttgart.

1848 ALEXANDER HALLEY, M.D., F.G.S., 7, Harley street, Cavendish square.

1819 †THOMAS HAMMERTON, 112, Piccadilly. C. 1829-30.

1838 HENRY HANCOCK, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; 37, Harley street, Cavendish square. C. 1851.

1849 *RICHARD JAMES HANSARD, late Surgeon to the Radcliffe Infirmary, Oxford.

1848 *GEORGE HARCOURT, M.D., Chertsey, Surrey.

1836 JOHN FOSSE HARDING, Mount Sandford, Southborough, Tunbridge Wells. C. 1858-9.

1856 CHARLES JOHN HARE, M.D., Professor of Clinical Medicine at University College, London; and Physician to University College Hospital; 41, Brook street, Grosvenor square.

1857 GEORGE HARLEY, M.D., F.C.S., Professor of Medical Jurisprudence in University College, London; and Assistant-Physician to University College Hospital; 77, Harley street, Cavendish square.

Elected

1859 Francis Harris, M.D., Assistant-Physician to, and Lecturer on Botany at, St. Bartholomew's Hospital; Assistant-Physician to the Hospital for Sick Children; 24, Cavendish square.

1843 Thomas Sunderland Harrison, M.D., F.L.S., 3, Portland place, Bath, Somersetshire.

1841 William Harvey, Surgeon to the Royal Dispensary for Diseases of the Ear, and to the Freemasons' Female Charity, and Aural Surgeon to the Great Northern Hospital; 2, Soho square. C. 1854.

1855 Alfred Haviland, Surgeon to the Bridgewater Infirmary; Bridgewater, Somerset.

1828 †Cæsar Henry Hawkins, F.R.S., Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.

1838 Charles Hawkins, Inspector of Anatomical Schools in London; Consulting Surgeon to Queen Charlotte's Hospital; 22, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Trans.* 2.

1848 Thomas Hawksley, M.D., Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 26, George street, Hanover square.

1860 Henry Howard Hayward, Assistant-Dental Surgeon to the Dental Hospital of London; Dental Surgeon to the Hospital for Consumption, Brompton; 56, Queen Anne street, Cavendish square.

1861 William Henry Hayward, Church House, Oldbury, near Birmingham.

1848 *James Newton Heale, M.D., Physician to the Winchester County Hospital; Winchester, Hants.

1850 George Heaton, M.D., Boston, U.S.

1829 †Thomas Heberden, M.D., 43, Park street, Grosvenor square.

1844 John Hennen, M.D. L. 1848-50.

1849 Amos Henriques, M.D., Hon. Physician to the Spanish Embassy; 67, Upper Berkeley street, Portman square.

Elected

1821 VINCENT HERBERSKI, M.D., Professor of Medicine in the University of Wilna.

1843 PRESCOTT GARDNER HEWETT, Surgeon to St. George's Hospital; 1, Chesterfield street, May fair. C. 1859. *Trans.* 7.

1855 GRAILY HEWITT, M.D., Physician to the British Lying-in Hospital; Assistant-Physician Accoucheur to, and Lecturer on Midwifery and the Diseases of Women and Children at, St. Mary's Hospital; 36, Berkeley square.

1853 THOMAS HEWLETT, Surgeon to Harrow School; Harrow, Middlesex. *Trans.* 1.

1841 *NATHANIEL HIGHMORE, Sherborne, Dorsetshire.

1862 MATTHEW BERKELEY HILL, M.B. Lond., Assistant-Surgeon to University College Hospital; 14, Weymouth street, Portland place.

1854 THOMAS HILLIER, M.D., Physician to the Hospital for Sick Children; Physician to the Skin Department of University College Hospital, and Medical Officer of Health for St. Pancras; 21, Upper Gower street.

1842 WILLIAM AUGUSTUS HILLMAN, Senior Assistant-Surgeon to the Westminster Hospital; 1, Argyll street, Regent street. C. 1858-9.

1841 †JOHN HILTON, F.R.S., *Vice-President*, Surgeon to Guy's Hospital; Consulting Surgeon to the Royal General Dispensary, St. Pancras; Examiner in Surgery at the University of London; 10, New Broad street, City. C. 1851. V.P. 1863-4. *Trans.* 3.

1859 FRANCIS HIRD, Assistant-Surgeon to, and Lecturer at, the Charing Cross Hospital; 13, Old Burlington street.

1840 THOMAS HODGKIN, M.D., Consulting Physician to the Hospital for Diseases of the Skin, and Member of the Senate of the University of London; 35, Bedford square. C. 1842-3. V.P. 1862-3. *Trans.* 6.

1813 JOSEPH HODGSON, F.R.S, 60, Westbourne terrace, Hyde park gardens. C. 1817. P. 1851-2. *Trans.* 1.

1861 *WILLIAM CARTER HOFFMEISTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Cowes, Isle of Wight.

Elected

1843 LUTHER HOLDEN, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Dispensary; Surgeon to the Foundling Hospital; 54, Gower street, Bedford square. C. 1859.

1814 †SIR HENRY HOLLAND, Bart., M.D., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen; 25, Brook street, Grosvenor square. C. 1817, 1833-4. V.P. 1826, 1840. *Trans.* 1.

1861 WILLIAM HENRY HOLMAN, M.B. Lond.; 68, Adelaide road south, Hampstead.

1856 TIMOTHY HOLMES, Assistant-Surgeon to, and Lecturer on Anatomy at, St. George's Hospital, and Surgeon to the Hospital for Sick Children; 22, Queen street, May fair. *Trans.* 2.

1846 BARNARD WIGHT HOLT, Senior Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3.

1846 CARSTEN HOLTHOUSE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the South London Ophthalmic Hospital; 2, Storey's gate, St. James's park. C. 1863.

1853 WILLIAM CHARLES HOOD, M.D., F.L.S., Visiting Physician in Lunacy to the Court of Chancery; Croydon Lodge, Surrey. *Trans.* 1.

1828 *EDWARD HOWELL, M.D., Senior Consulting Physician to the Swansea Infirmary; 2, South Hill place, Swansea, Glamorganshire.

1857 JOHN WHITAKER HULKE, Assistant-Surgeon to the Middlesex Hospital, and to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. *Trans.* 3.

1857 EDWARD CHARLES HULME, Surgeon to the Great Northern Hospital; Surgeon to the Central London Ophthalmic Hospital; 19, Gower street, Bedford square. *Trans.* 1.

1844 EDWIN HUMBY, 83, Hamilton terrace, St. John's wood.

Elected

1855 GEORGE MURRAY HUMPHRY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital, and Lecturer on Anatomy in the Cambridge University Medical School; Cambridge. *Trans.* 4.

1849 EDWARD LAW HUSSEY, Surgeon to the Radcliffe Infirmary; 104, St. Aldate's, Oxford. *Trans.* 1.

1856 JONATHAN HUTCHINSON, Surgeon to, and Lecturer on Surgery at, the London Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Surgeon to the Metropolitan Free Hospital; 4, Finsbury circus. *Pro.* 2.

1820 WILLIAM HUTCHINSON, M.D.

1840 CHARLES HUTTON, M.D., Senior Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.

1847 WILLIAM EDMUND IMAGE, Senior Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.

1856 CORNELIUS INGLIS, M.D.

1826 WILLIAM INGRAM, Midhurst, Sussex.

1845 *HENRY JACKSON, Senior Surgeon to the Sheffield General Infirmary; St. James's row, Sheffield, Yorkshire.

1841 PAUL JACKSON, 24, Wimpole street, Cavendish square. C. 1862.

1863 THOMAS VINCENT JACKSON, Surgeon to the South Staffordshire General Hospital; Darlington street, Wolverhampton.

1841 MAXIMILIEN MORRIS JACOBOVICS, M.D., Vienna.

1825 JOHN B. JAMES, M.D

1847 *WILLIAM WITHALL JAMES, Surgeon to the Devon and Exeter Hospital; Exeter, Devonshire.

1844 SAMUEL JOHN JEAFFRESON, M.D., Physician to the Warneford Hospital, and Warwick Dispensary; Leamington, Warwickshire.

1839 JULIUS JEFFREYS, F.R.S., Drymona, Belvidere road south, Upper Norwood, Surrey.

1840 *GEORGE SAMUEL JENKS, M.D., 18, Circus, Bath.

Elected

1851 WILLIAM JENNER, M.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Physician to University College Hospital, and Professor of the Principles and Practice of Medicine at University College; 8, Harley street, Cavendish square. C. 1864. *Trans.* 3.

1848 ATHOL ARCHIBALD WOOD JOHNSON, 20, Regency square, Brighton. *Trans.* 1.

1851 EDMUND CHARLES JOHNSON, M.D., Corresponding Member of the Medical and Philosophical Society of Florence and of "L'Institut Genevois;" C., No. 3, Albany, Piccadilly.

1847 GEORGE JOHNSON, M.D., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. *Trans.* 5.

1862 CHARLES HANDFIELD JONES, M.B., F.R.S., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; 49, Green street, Grosvenor square.

1844 †HENRY BENCE JONES, M.A., M.D., F.R.S.; 31, Brook street, Grosvenor square. C. 1855-6. *Trans.* 11.

1835 HENRY DERVICHE JONES, 12, Norfolk crescent, Oxford square. C. 1854-5.

1837 THOMAS WILLIAM JONES, M.D., 19, Finsbury pavement, and Green street, Ponders End. C. 1858.

1859 WILLIAM PRICE JONES, M.D., Surbiton, Kingston.

1829 *GEORGE CHARLES JULIUS, Richmond, Surrey.

1816 *GEORGE HERMANN KAUFFMAN, M.D., Hanover.

1848 *DANIEL BURTON KENDELL, M.D., Kettlethorpe Hall, Wakefield, Yorkshire.

1847 ALFRED KEYSER, 21, Norfolk crescent, Oxford square.

1857 HENRY WALTER KIALLMARK, late Staff Surgeon, 2nd class, attached to the Ottoman Army; 46, Prince's square, Westbourne grove.

1839 *DAVID KING, M.D., Medical Officer of Health for Eltham; Eltham, Kent.

1851 JOHN ABERNETHY KINGDON, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, City. *Trans.* 1.

Elected

1858 WILLIAM SENHOUSE KIRKES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 2, Lower Seymour street, Portman square. *Trans.* 1.

1855 JAMES ROBERT LANE, Surgeon to, and Lecturer on Operative Surgery at, St. Mary's Hospital, and Surgeon to the Lock and St. Mark's Hospitals, 1, Grosvenor place.

1840 SAMUEL ARMSTRONG LANE, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital, and Consulting Surgeon to the Lock Hospital; 1, Grosvenor place. C. 1849-50.

1841 *CHARLES LASHMAR, M.D., 83, North End, Croydon, Surrey.

1862 PETER WALLWORK LATHAM, M.A., M.B., Physician to Addenbrooke's Hospital, Cambridge; Examiner for Medical Degrees in Cambridge University; 15, Sidney street, Cambridge.

1816 G. E. LAWRENCE.

1809 †WILLIAM LAWRENCE, F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon to, and Lecturer on Surgery, at St. Bartholomew's Hospital, and Surgeon to Bridewell and Bethlem Hospital; Foreign Associate of the Imperial Academy of Medicine of Paris; 18, Whitehall place. S. 1813-7. V.P. 1818-9. T. 1821-6. P. 1831-2. C. 1820, 1833-4, 1842-3. *Trans.* 18.

1840 THOMAS LAYCOCK, M.D., F.R.S.E., Professor of the Practice of Medicine and of Clinical Medicine, and Lecturer on Psychology and Mental Diseases in the University of Edinburgh, and Physician to the Edinburgh Royal Infirmary; 4, Rutland street, Edinburgh.

1843 *JESSE LEACH, Moss Hall, Heywood, near Bury, Lancashire.

1822 JOHN JOSEPH LEDSAM, M.D., 17, Esplanade, Scarborough, Yorkshire.

1823 †HENRY LEE, M.D., Weather Oak, Alvechurch, near Bromsgrove, Worcestershire. C. 1837. S. 1839-40.

Elected

1843 HENRY LEE, *Librarian*, Surgeon to, and Lecturer on Pathology at, St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. *Trans.* 7. *Pro.* 1.

1822 †ROBERT LEE, M.D., F.R.S., Lecturer on Midwifery at St. George's Hospital; Corresponding Member of the Imperial Academy of Medicine, Paris; 4, Savile row, Burlington gardens. C. 1829, 1834. S. 1830-3. V.P. 1835. *Trans.* 26.

1836 FREDERICK LEIGHTON, M.D., Frankfort-on-the-Maine.

1854 HANANEL DE LEON, M.D., 15, Holland villas road, North Kensington.

1806 JOHN LIND, M.D.

1845 WILLIAM JOHN LITTLE, M.D., 34, Brook street, Grosvenor square.

1819 ROBERT LLOYD, M.D.

1820 J. G. LOCHER, M.C.D., Town Physician of Zurich. *Trans.* 2.

1824 †SIR CHARLES LOCOCK, Bart., M.D., F.R.S., First Physician Accoucheur to H.M. the Queen; Member of the Senate of the University of London; 26, Hertford street, Mayfair. C. 1826. V.P. 1841. P. 1857-8. *Trans.* 1.

1852 CHARLES LODGE, M.D., "United States Army."

1846 HENRY THOMAS LOMAX, Surgeon to the County Police; St. Mary's grove, Stafford.

1860 THOMAS LONGMORE, Deputy Inspector-General and Professor of Clinical and Military Surgery; New Army Medical School, Royal Victoria Hospital, Netley, Southampton. *Trans.* 1.

1836 JOSEPH S. LÖWENFELD, M.D., Berbice.

1852 JAMES LUKE, F.R.S., Consulting Surgeon to the London Hospital, and Surgeon to St. Luke's Hospital for Lunatics; Woolley Lodge, Maidenhead Thicket, Berks. C. 1858. *Trans.* 4.

1857 FELIX WILLIAM LYON, M.D., Lyndhurst, Hants.

1862 *ROBERT M'DONNELL, M.D., Examiner in Anatomy and Physiology, Queen's University in Ireland; 11, Lower Pembroke street, Dublin.

1846 WILLIAM M'EWEN, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.

Elected

1823 †GEORGE MACILWAIN, Consulting Surgeon to the Finsbury Dispensary and the St. Ann's Society's Schools; 3, the Court yard, Albany. C. 1829-30. V.P. 1848. *Trans.* 1.

1822 RICHARD MACINTOSH, M.D.

1859 *JOHN M'INTYRE, M.D., Odiham, Hants.

1848 FREDERICK WILLIAM MACKENZIE, M.D., Physician to Queen Charlotte's Lying-in Hospital; 11, Chester place, Hyde park square. *Trans.* 2.

1818 WILLIAM MACKENZIE, M.D., Surgeon-Oculist to H.M. the Queen in Scotland, and Surgeon to the Glasgow Eye Infirmary; 49, Bath street, Glasgow. *Trans.* 2.

1854 *DRAPER MACKINDER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1844 DANIEL MACLACHLAN, M.D., late Physician to the Royal Hospital, Chelsea; Deputy Inspector-General of Hospitals. C. 1860-1. *Trans.* 1.

1860 JOHN MACLEAN, M.D., 24, Portman street, Portman square.

1849 DUNCAN MACLACHLAN MACLURE, 16, Harley street, Cavendish square.

1842 JOHN MACNAUGHT, M.D., Bedford street, Liverpool.

1837 ANDREW MELVILLE M'WHINNIE, 7, Serjeant's Inn, Temple. C. 1851-2. *Trans.* 1.

1855 WILLIAM MARCET, M.D., F.R.S., 1, Torrington street, Torrington square. *Trans.* 1.

1848 WILLIAM ORLANDO MARKHAM, M.D., Physician to St. Mary's Hospital; 33, Clarges street, Piccadilly. C. 1862-3. *Trans.* 2.

1838 THOMAS PARR MARSH, M.D., Consulting Physician to the Salop Infirmary, Shrewsbury; Coed Cefer, Monmouth.

1851 JOHN MARSHALL, F.R.S., Surgeon to University College Hospital; Fullerian Professor of Physiology at the Royal Institution of Great Britain; 10, Savile row, Burlington gardens. *Trans.* 2.

1841 SIR JAMES RANALD MARTIN, C.B., F.R.S., Examining Medical Officer to the Secretary of State for India in Council; 37, Upper Brook street. C. 1853. V.P. 1862.

Elected

1849 GEORGE BELLASIS MASFEN, Ghazeepore, India.

1853 WILLIAM EDWARD MASFEN, Surgeon to the Staffordshire General Infirmary; Stafford.

1864 FRANCIS MASON, Assistant-Surgeon to King's College Hospital; 10, Conduit street, Regent street.

1837 THOMAS MAYO, M.D., F.R.S.; the Refuge, Yarmouth, Isle of Wight. S. 1841. C. 1847-8. V.P. 1851-2.

1839 RICHARD HENRY MEADE, Senior Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1837 SAMUEL WILLIAM JOHN MERRIMAN, M.D., Consulting Physician-Accoucheur to the Westminster General Dispensary, and Physician-Accoucheur to the Western General Dispensary; 3, Charles street, Westbourne terrace. C. 1851-2. *Trans.* 1.

1852 JAMES MERRYWEATHER, Consulting Surgeon to the National Dental Hospital; 57, Brook street, Grosvenor square.

1847 EDWARD MERYON, M.D., F.G.S., 14, Clarges street, Piccadilly. L. 1859-60. C. 1864. *Trans.* 1.

1815 AUGUSTUS MEYER, M.D., St. Petersburgh.

1840 RICHARD MIDDLEMORE, Consulting Surgeon to the Birmingham Eye Infirmary; Temple row, Birmingham.

1854 EDWARD ARCHIBALD MIDDLESHIP, late of Richmond, Surrey.

1860 *HERBERT CHALMERS MILES, Assistant-Surgeon in the Royal Artillery, Woolwich.

1818 *PATRICK MILLER, M.D., F.R.S.E., Senior Physician to the Devon and Exeter Hospital, and to St. Thomas's Hospital for Lunatics; the Grove, Exeter, Devonshire.

1863 HENRY MONRO, M.D., Physician to St. Luke's Hospital, 13, Cavendish square.

1844 NATHANIEL MONTEFIORE, 36, Hyde park gardens.

1848 CHARLES HEWITT MOORE, Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital; 102, Piccadilly. L. 1858. S. 1859-62. C. 1864. *Trans.* 7.

1836 GEORGE MOORE, M.D., Hastings, Sussex.

Elected

1861 CHARLES MOREHEAD, M.D., Hon. Surgeon to H.M. the Queen; Deputy-Inspector General of Hospitals; late Principal of Grant Medical College, Bombay, &c.

1857 JOHN MORGAN, 3, Sussex place, Hyde park gardens.

1861 JOHN EDWARD MORGAN, M.B., Lecturer on Pathology at the Manchester Royal School of Medicine; 3, Gore street, Piccadilly, Manchester.

1851 FREDERIC JOHN MOUAT, M.D., Professor of Medicine in the Medical College of Calcutta, Secretary of the Council of Education in India, and Inspector-General of Gaols, Lower Provinces; Calcutta.

1856 CHARLES MURCHISON, M.D., Senior Physician to the London Fever Hospital; Assistant-Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; 79, Wimpole street, Cavendish square. *Trans.* 3.

1847 SIMON MURCHISON, Bicester, Oxon.

1863 ARTHUR B. R. Myers, Coldstream Guards Hospital, Vincent square, Westminster.

1859 GEORGE NAYLER, Assistant-Surgeon to the Royal Orthopædic Hospital, 8, George street, Hanover square.

1835 THOMAS ANDREW NELSON, M.D., 10, Nottingham terrace, York gate, Regent's park.

1843 EDWARD NEWTON, 30, Fitzroy square. C. 1863-4.

1849 HENRY BURFORD NORMAN, Portland Lodge, Southsea, Hants.

1845 HENRY NORRIS, Charmouth, Dorset.

1847 *WILLIAM EDWARD CHARLES NOURSE, Surgeon to St. Mary's Hospital, Brighton; 11, Marlborough place, Brighton.

1849 ARTHUR NOVERRE, 25, South street, Park lane.

1864 THOMAS WILLIAM NUNN, Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1859 *THOMAS NUNNELEY, Senior Surgeon to the Leeds Eye and Ear Infirmary; Leeds. *Trans.* 2

1847 THOMAS O'CONNOR, March, Cambridgeshire.

Elected

1843 WILLIAM O'CONNOR, M.D., Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.

1858 JOHN WILLIAM OGLE, M.D., Assistant-Physician to, and Lecturer on Pathology at, St. George's Hospital; 13, Upper Brook street, Grosvenor square. *Trans.* 4.

1855 *WILLIAM OGLE, M.A., M.D., Physician to the Derby Infirmary; 3, Stewart terrace, Derby.

1860 WILLIAM OGLE, M.D., Lecturer on Physiology at St. George's Hospital, and Physician to the St. George's and St. James's Dispensary, 37, Clarges street, Piccadilly.

1850 HENRY OLDHAM, M.D., Obstetric Physician to, and Clinical Lecturer on Midwifery at, Guy's Hospital; 26, Finsbury square. *Trans.* 1.

1846 *EDWARD LATHAM ORMEROD, M.D., Physician to the Sussex County Hospital; 14, Old Steine, Brighton. *Trans.* 2.

1847 *WILLIAM BOUSFIELD PAGE, Surgeon to the Cumberland Infirmary; Carlisle. *Trans.* 2.

1840 JAMES PAGET, F.R.S., Surgeon Extraordinary to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Surgeon to St. Bartholomew's and Christ's Hospitals, and Member of the Senate of the University of London; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. *Trans.* 8.

1858 *WILLIAM PALEY, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.

1861 JAMES PALFREY, M.D., Senior Assistant-Physician to the Metropolitan Free Hospital; Physician to the Surrey Dispensary; 12, Wellington street, Southwark.

1836 LANGSTON PARKER, Hon. Surgeon to the Queen's Hospital, Birmingham; Colmore row, Birmingham.

1847 NICHOLAS PARKER, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 22, Finsbury square.

1841 JOHN PARKIN, M.D., Rome.

1851 JAMES PART, M.D., 7, Camden road villas, Camden town.

Elected

1828 †Richard Partridge, F.R.S., *President*, Professor of Anatomy to the Royal Academy of Arts, Surgeon to King's College Hospital, and Professor of Anatomy in King's College, London; 17, New street, Spring gardens. S. 1832-6. C. 1837-8. V.P. 1847-8. C. 1861-2. P. 1863-4.

1845 Thomas Bevill Peacock, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria park; 20, Finsbury circus. S. 1855-6. *Trans.* 2.

1864 David Ritchie Pearson, M.D., 23, Upper Phillimore place, Kensington.

1856 Richard King Peirce, 16, Norland place, Notting hill.

1830 Charles P. Pelechin, M.D., St. Petersburgh.

1855 *Oliver Pemberton, Surgeon to the Birmingham General Hospital, and Lecturer on Surgical Pathology at Sydenham College; 18, Temple row, Birmingham. *Trans.* 1.

1844 William Vesalius Pettigrew, M.D., Surgeon to the Female Orphan Asylum, Lambeth; 7, Chester street, Grosvenor place.

1848 Edward Phillips, M.D., F.L.S., Physician to the Coventry and Warwickshire Hospital; Coventry, Warwickshire.

1852 Richard Phillips, 50, Leinster square, Westbourne grove.

1854 Thomas Bacon Phillips, M.D., Physician to the Brighton and Hove Dispensary; 36, Lansdowne place, Brighton.

1846 Francis Richard Philp, M.D., Colby House, Kensington, and Sherborne House, Harrogate, Yorkshire.

1851 *James Hollins Pickford, M.D., M.R.I.A., 1, Cavendish place, Brighton.

1836 Isaac Pidduck, M.D., Physician to the Bloomsbury Dispensary; 22, Montague street, Russell square. *Pro.* 2.

1841 Henry Alfred Pitman, M.D., *Treasurer*, Physician to, and Lecturer on Medicine at, St. George's Hospital; Consulting Physician to the Royal General Dispensary, St. Pancras; 94, Gloucester place, Portman square, L. 1851-3. C. 1861-2. T. 1863-4.

Elected

1850 ALFRED POLAND, Surgeon to, and Lecturer on Surgery at, Guy's Hospital and to the Eye Infirmary attached to the Hospital; 58, Welbeck street, Cavendish square.

1845 GEORGE DAVID POLLOCK, Surgeon in Ordinary to H.R.H. the Prince of Wales; Surgeon to St. George's Hospital; 27, Grosvenor street. C. 1856-7. L. 1859-62. *Trans.* 2.

1843 CHARLES POPE, M.D., Glastonbury, Somersetshire.

1846 JEPHSON POTTER, M.D., F.L.S., Physician to the Liverpool General Hospital for Consumption and Diseases of the Chest; 109, Upper Parliament street, Liverpool.

1842 JAMES POWELL, M.B.

1851 ROBERT FRANCIS POWER, M.D., 71, Gloucester place, Portman square.

1857 WILLIAM OVEREND PRIESTLEY, M.D., Physician-Accoucheur to H.R.H. the Princess Louis of Hesse; Professor of Midwifery in King's College, London; and Physician for the Diseases of Women and Children to King's College Hospital; Consulting Physician-Accoucheur to the St. Marylebone Infirmary; 17, Hertford street, May fair.

1839 JOHN PROPERT, Consulting Surgeon to the Society of Ancient Britons; 6, New Cavendish street, Portland place.

1845 JOHN PYLE, 56, Oxford terrace, Hyde park. C. 1860.

1830 JONES QUAIN, M.D., Paris.

1850 RICHARD QUAIN, M.D., Physician to the Hospital for Consumption and Diseases of the Chest, and Member of the Senate of the University of London; 56, Harley street, Cavendish square. *Trans.* 1.

1835 †RICHARD QUAIN, F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Consulting Surgeon to the Eye Infirmary attached to the Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Trans.* 1. *Pro.* 2.

Elected

1852 CHARLES BLAND RADCLIFFE, M.D., Physician to, and Lecturer on Materia Medica at, the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square.

1857 HENRY RANKE, M.D., Munich.

1854 WILLIAM HENRY RANSOM, M.D., Physician to the Nottingham General Hospital; the Pavement, Nottingham.

1859 HENRY HUNTER RAYMOND, Cirencester, Gloucestershire.

1858 FREDERICK GEORGE REED, M.D., 46, Hertford street, May fair. *Trans.* 1.

1821 HENRY REEDER, M.D.

1857 GEORGE OWEN REES, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 26, Albemarle street, Piccadilly. *Trans.* 1.

1855 JOHN RUSSELL REYNOLDS, M.D., Professor of Clinical Medicine at University College, London, and Physician to University College Hospital; 38, Grosvenor street.

1847 SAMUEL RICHARDS, M.D., 36, Bedford square.

1852 CHRISTOPHER THOMAS RICHARDSON, M.B.

1829 SIR JOHN RICHARDSON, Knt., M.D., LL.D., C.B., F.R.S., late Inspector General of Hospitals and Fleets; Lancrigg, Grasmere, Westmoreland.

1849 *WILLIAM RICHARDSON, M.D., 9, Ephraim road, Tunbridge Wells, Kent.

1845 BENJAMIN RIDGE, M.D., 21, Bruton street, Berkeley square.

1843 JOSEPH RIDGE, M.D., 39, Dorset square. C. 1858. *Pro.* 1.

1852 CHARLES RIDLEY, Surgeon to the Royal Society for Protection of Life from Fire; 6, Charlotte street, Bedford square.

1863 SYDNEY RINGER, M.D., Professor of Materia Medica at University College, and Assistant-Physician to University College Hospital; Assistant-Physician to the Hospital for Sick Children; 29, Welbeck street, Cavendish square.

1864 CHARLES GEORGE RITCHIE, M.D., 36, Mount street, Grosvenor square.

1852 JOHN ROBERTS, M.R.C.P., 75, Grosvenor street.

Elected

1855 CHARLES ALEXANDER LOCKHART ROBERTSON, M.D., Medical Superintendent of the Sussex County Lunatic Asylum; Hayward's Heath, Sussex.

1857 JOHN CHARLES GEORGE ROBERTSON, Assistant Medical Officer, Female Department, Middlesex Lunatic Asylum, Hanwell.

1862 CHARLES ROBINSON, 11, Montagu street, Montagu square.

1843 GEORGE ROBINSON, M.D., 26, Welbeck street, Cavendish square. *Trans.* 2.

1843 WILLIAM RODEN, M.D., the Grange, Kidderminster, Worcestershire.

1835 GEORGE HAMILTON ROE, M.D., Senior Physician to the Hospital for Consumption and Diseases of the Chest; 57, Park street, Grosvenor square. C. 1841-2. *Trans.* 1.

1836 ARNOLD ROGERS, Consulting Surgeon-Dentist to St. Bartholomew's Hospital; 16, Hanover square.

1829 WILLIAM SUDLOW ROOTS, F.L.S., Surgeon to the Royal Establishment at Hampton Court; Kingston, Surrey.

1850 GEORGE ROPER, 168, Shoreditch.

1855 THOMAS TATTERSALL ROSCOW, M.D.

1836 *CALEB BURRELL ROSE, F.G.S., 25, King street, Great Yarmouth, Norfolk. *Trans.* 1.

1857 HENRY COOPER ROSE, M.D., High street, Hampstead.

1849 CHARLES HENRY FELIX ROUTH, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Trans.* 1.

1863 THOMAS SMITH ROWE, M.D., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1834 HENRY WYLDBORE RUMSEY, Wolseley house, Cheltenham.

1845 JAMES RUSSELL, M.D., Physician to the Birmingham General Hospital, and Lecturer on Pathology and Therapeutics at Sydenham College; 91, New Hall street, Birmingham.

1851 HENRY HYDE SALTER, M.D., F.R.S.; Assistant-Physician to, and Lecturer on Physiology and Pathology at, the Charing Cross Hospital; 6, Montague street, Russell square.

Elected

1856 SAMUEL JAMES A. SALTER, F.R.S., F.L.S., Surgeon-Dentist to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City. *Trans.* 2.

1849 HUGH JAMES SANDERSON, M.D., Physician to the Hospital for Women; 26, Upper Berkeley street, Portman square.

1855 JOHN BURDON SANDERSON, M.D., Assistant-Physician to the Middlesex Hospital; Medical Officer of Health for Paddington; 49, Queen Anne street, Cavendish square.

1847 WILLIAM HENRY OCTAVIUS SANKEY, M.D., Sandywell park, near Cheltenham.

1845 EDWIN SAUNDERS, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square.

1834 LUDWIG V. SAUVAN, M.D, Warsaw.

1859 WILLIAM SCOVELL SAVORY, F.R.S., Assistant-Surgeon to, and Lecturer on General Anatomy and Physiology at, St. Bartholomew's Hospital; Examiner in Physiology and Comparative Anatomy at the University of London; 23A, Brook street, Grosvenor square. *Trans.* 3.

1853 MAURICE SCHULHOF, M.D., Physician to the Royal General Dispensary, Bartholomew Close; 14, Brook street, Grosvenor square.

1861 *WILLIAM SCOTT, M.D., Physician to the Huddersfield Infirmary; 12, New North road, Huddersfield.

1858 *GEORGE SCRATCHLEY, M.D., New Orleans, Louisiana, U.S.

1863 WILLIAM SEDGWICK, Surgeon to the St. Marylebone Provident Dispensary; 12, Park place, Upper Baker street.

1856 EDWIN SERCOMBE, Surgeon-Dentist to St. Mary's Hospital; 49, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1824 †EDWARD JAMES SEYMOUR, M.D., F.R.S., Consulting Physician to the Seamen's Hospital Ship 'Dreadnought'; 13, Charles street, Berkeley square. C. 1826, 1831. S. 1827-8. V.P. 1830, 1842. *Trans.* 2.

1840 WILLIAM SHARP, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1837 †WILLIAM SHARPEY, M.D., F.R.S., LL.D., Professor of Anatomy and Physiology in University College, London; Member of the Senate of the University of London, and Secretary of the Royal Society; 33, Woburn place, Russell square. C. 1848-9. V.P. 1862.

Elected

1836 †ALEXANDER SHAW, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 25, Henrietta street, Cavendish square. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Trans.* 4.

1848 *EDWARD JAMES SHEARMAN, M.D., Rotherham, Yorkshire.

1859 SEPTIMUS WILLIAM SIBLEY, Lecturer on Pathological Anatomy at the Middlesex Hospital; 12, New Burlington street. *Trans.* 3.

1849 FRANCIS SIBSON, M.D., F.R.S., Physician to St. Mary's Hospital; Examiner in the Practice of Medicine at the University of London; 40, Brook street, Grosvenor square. C. 1863-4. *Trans.* 1.

1848 EDWARD HENRY SIEVEKING, M.D., Physician in Ordinary to H.R.H. the Prince of Wales; Physician to, and Lecturer on Materia Medica at, St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. *Trans.* 2.

1842 JOHN SIMON, F.R.S., Surgeon to, and Lecturer on General Pathology at, St. Thomas's Hospital; Medical Officer of the Privy Council; 8, Richmond terrace. C. 1854-55. *Trans.* 1.

1857 JAMES LEWIS SIORDET, M.B., Mentone.

1827 GEORGE ROBERT SKENE, Bedford.

1824 †FREDERIC CARPENTER SKEY, F.R.S., Consulting Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; 13, Grosvenor street. C. 1828. L. 1829-31. V.P. 1841-2. P. 1859-60. *Trans.* 1.

1852 CHARLES CASE SMITH, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk.

1835 JOHN GREGORY SMITH, Harewood, Leeds, Yorkshire.

1843 ROBERT WILLIAM SMITH, M.D., M.R.I.A., Professor of Surgery in the University of Dublin; Surgeon to the Richmond Hospital; 63, Eccles street, Dublin.

1838 SPENCER SMITH, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; 9, Queen Anne street, Cavendish square. C. 1854. S. 1855-8. V.P. 1859-60.

Elected

1863 THOMAS SMITH, Assistant-Surgeon to, and Demonstrator of Anatomy at, St. Bartholomew's Hospital, and Assistant-Surgeon to the Hospital for Sick Children; 7, Montague street, Russell square.

1864 *THOMAS HECKSTALL SMITH, Rowlands St. Mary Cray, Kent.

1845 WILLIAM SMITH, Surgeon to the Chesterfield and North Derbyshire Hospital and Dispensary, Chesterfield, Derbyshire. *Trans.* 1.

1847 WILLIAM SMITH, M.D., Consulting Physician to the Weymouth Infirmary; Weymouth, Dorsetshire.

1850 WILLIAM TYLER SMITH, M.D., Examiner in Midwifery at the University of London; Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 21, Upper Grosvenor street. *Trans.* 2.

1851 JOHN SODEN, Surgeon to the Bath United Hospital, and Consulting Surgeon to the Bath Eye Infirmary; 24, Circus, Bath. *Trans.* 2.

1830 †SAMUEL SOLLY, F.R.S., Surgeon to St. Thomas's Hospital; Consulting Surgeon to the Royal General Dispensary, Bartholomew Close: 6, Savile row. L. 1838-40. C. 1845-6. V.P. 1849-50. *Trans.* 6.

1844 FREDERICK R. SPACKMAN, M.D., Harpenden, St. Alban's.

1834 JAMES SPARK, Italy.

1851 ROBERT JOHN SPITTA, M.B., Medical Officer to the Clapham General Dispensary; Clapham, Surrey. *Trans.* 1.

1843 *STEPHEN SPRANGER, Hursley, Hampshire.

1857 JOHN STANTON, M.D., 9, Montagu square.

1851 JAMES STARTIN, Surgeon to, and Lecturer on Cutaneous Disorders at, the Hospital for Diseases of the Skin, Blackfriars; 3, Savile row, Burlington gardens.

1854 HENRY STEVENS, M.D., Lond., 78, Grosvenor street.

1842 ALEXANDER PATRICK STEWART, M.D., *Librarian*, Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 74, Grosvenor street. C. 1856-7. L. 1863-4.

1859 WILLIAM EDWARD STEWART, 12, Weymouth st., Portland pl.

1856 ALONZO HENRY STOCKER, M.D., Resident Medical Superintendent of Grove Hall Lunatic Asylum, Bow.

1843 ROBERT REEVE STORKS, Paris.

Elected

1858 †JOHN FREMLYN STREATFEILD, Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and Assistant-Surgeon to the Eye Infirmary attached to University College Hospital; 15, Upper Brook street, Grosvenor square.

1863 OCTAVIUS STURGES, M.B., Physician to the Chelsea, Brompton, and Belgrave Dispensary; 35, Connaught square.

1839 ALEXANDER JOHN SUTHERLAND, M.D., F.R.S., Consulting Physician to the Royal South London Dispensary; 6, Richmond terrace, Whitehall. C. 1850-1. V.P. 1858-9. *Trans.* 1.

1860 SIGISMUND SUTRO, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

1855 JOHN MAULE SUTTON, M.D., Bloomfield, Narberth, Pembrokeshire.

1861 *GEORGE BACON SWEETING, King's Lynn, Norfolk.

1842 JAMES SYME, F.R.S.E., Surgeon in Ordinary to H.M. the Queen in Scotland; Professor of Clinical Surgery in the University of Edinburgh, and Surgeon to the Edinburgh Royal Infirmary; 2, Rutland street, Edinburgh. *Trans.* 5.

1854 *FREDERICK SYMONDS, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 32, Beaumont street, Oxford.

1844 RICHARD WILLIAM TAMPLIN, Surgeon to the Royal Orthopædic Hospital; 33, Old Burlington street.

1848 THOMAS HAWKES TANNER, M.D., F.L.S., 9, Henrietta street, Cavendish square.

1864 GABRIEL TAUSSIG, M.D., 70, Piazza Barberini, Rome.

1852 ROBERT TAYLOR, Surgeon to the Central London Ophthalmic Hospital, and to the Cripple's Home, Hill street; 21, Edwards street, Portman square.

1845 THOMAS TAYLOR, Lecturer on Chemistry at the Middlesex Hospital Medical School; 4, Vere street, Cavendish square.

Elected

1856 THOMAS PRIDGIN TEALE, F.R.S., F.L.S., Surgeon to the Leeds General Infirmary; Albion street, Leeds. *Trans.* 2.

1859 EDWARD TEGART, Junior, 49, Jermyn street, St. James's.

1862 EDMUND SYMES THOMPSON, M.D., Assistant-Physician to King's College Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 3, Upper George street, Portman square.

1857 HENRY THOMPSON, M.D., Physician to, and Lecturer on Materia Medica at, the Middlesex Hospital; 52, Welbeck street, Cavendish square.

1852 HENRY THOMPSON, Surgeon Extraordinary to H.M. the King of the Belgians, Surgeon to University College Hospital, and Consulting Surgeon to the St. Marylebone Infirmary; 35, Wimpole street, Cavendish square. *Trans.* 3.

1862 REGINALD EDWARD THOMPSON, M.D., Physician to the St. George's and St. James's Dispensary; 6, Grosvenor street, Grosvenor square.

1836 JOHN THURNAM, M.D., Resident Medical Superintendent of the Wilts County Asylum, Devizes, Wiltshire. *Trans.* 4.

1848 EDWARD JOHN TILT, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 60, Grosvenor street.

1828 JAMES TORRIE, M.D., Old Bridge of Don, by Aberdeen.

1843 JOSEPH TOYNBEE, F.R.S., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital; Consulting Aural Surgeon to the Asylum for the Deaf and Dumb, and to the St. George's and St. James's Dispensary; 18, Savile row, Burlington gardens. C. 1863-4. *Trans.* 9. *Pro.* 1.

1850 SAMUEL JOHN TRACY, Surgeon-Dentist to St. Bartholomew's and Christ's Hospitals; 28, Old Burlington street.

1859 EDWIN THOMAS TRUMAN, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1864 THOMAS JOLLIFFE TUFNELL, Examiner in Surgery to the Royal College of Surgeons of Ireland; 58, Lower Mount street, Merrion square, Dublin.

Elected

1862 THOMAS HARRINGTON TUKE, M.D., Manor house, Chiswick, and 37, Albemarle street.

1855 JAMES STEWART TULLOCH, M.D., 1, Pembridge place, Bayswater.

1845 THOMAS TURNER, F.L.S., Consulting Surgeon to the Manchester Royal Infirmary, and Lecturer on Anatomy and Physiology at the Manchester Royal School of Medicine; 77, Mosley street, Manchester.

1846 ALEXANDER URE, Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital, and Consulting Surgeon to the Westminster General Dispensary; 18, Upper Seymour street, Portman square. *Trans.* 1.

1806 BOWYER VAUX, Teignmouth, Devon.

1828 BENEDETTO VULPES, M.D., Physician to the Hospital of Aversa, and to the Hospital of Incurables, Naples.

1854 EDWARD WADDINGTON, Kettlethorpe Hall, Newark, Notts.

1841 ROBERT WADE, Senior Surgeon to the Westminster General Dispensary; 68, Dean street, Soho. *Trans.* 1.

1864 CHARLES DERBY WAITE, M.B., 30, Old Burlington street.

1861 *JAMES WALSH, M.D., Staff-Surgeon, R.N., 41, Cathrine street, Limerick, Ireland.

1852 WALTER HAYLE WALSHE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 37, Queen Anne street, Cavendish square. *Trans.* 1.

1851 HENRY HAYNES WALTON, Surgeon to the Central London Ophthalmic Hospital, and Surgeon to St. Mary's Hospital; 69, Brook street, Hanover sq. *Trans.* 1. *Pro.* 1.

1852 DANIEL WANE, M.D., 20, Grafton street, Berkeley square.

1821 WILLIAM TILLEARD WARD.

1858 JOHN RICHARD WARDELL, M.D., 4, Belmont, Tunbridge Wells.

1846 JAMES THOMAS WARE, Consulting Surgeon to the Finsbury Dispensary, and Hon. Surgeon to the Metropolitan Convalescent Institution; 18, Gordon square.

1818 JOHN WARE, Clifton Down, near Bristol.

1814 †MARTIN WARE, 18, Gordon square. C. 1844-5. T. 1846. V.P. 1853.

Elected

1829 ELIAS TAYLOR WARRY, M.D., Yeovil, Somerset.

1861 A. T. HOUGHTON WATERS, M.D., Physician to the Liverpool Northern Hospital, and Lecturer on Anatomy and Physiology in the Liverpool Royal Infirmary School of Medicine; 27, Hope Street, Liverpool. *Trans.* 2.

1837 THOMAS WATSON, M.D., F.R.S., D.C.L., President of the Royal College of Physicians; Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6.

1861 WILLIAM SPENCER WATSON, M.B., Assistant-Surgeon to King's College Hospital; 27, Montague street, Russell square.

1854 WILLIAM WEBB, M.D., Wirksworth, Derbyshire.

1840 WILLIAM WOODHAM WEBB, M.D., Cliff House, Kirtley, South Lowestoft, Suffolk.

1842 FREDERIC WEBER, M.D., 44, Green street, Park lane. C. 1857.

1857 HERMANN WEBER, M.D., Physician to the German Hospital; 49, Finsbury square. *Trans.* 3.

1835 †JOHN WEBSTER, M.D., F.R.S., Physician to the Scottish Hospital, and Consulting Physician to the St. George's and St. James's Dispensary; 20, Brook street, Grosvenor square. C. 1843-4. V.P. 1855-6. *Trans.* 6. *Pro.* 1.

1844 WILLIAM WEGG, M.D., Physician to the St. George's and St. James's Dispensary; 26, Park lane. L. 1854-8. C. 1861-2.

1861 JOHN SOELBERG WELLS, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; 16, Savile row.

1854 THOMAS SPENCER WELLS, Surgeon in Ordinary, to H.M.'s Household; Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. *Trans.* 3. *Pro.* 1.

1842 †CHARLES WEST, M.D., Examiner in Midwifery at the University of London, and Physician to the Hospital for Sick Children; 61, Wimpole street, Cavendish square. C. 1855-6. V.P. 1863. *Trans.* 2.

Elected

1841 THOMAS WEST, M.D., Daventry, Northamptonshire.

1828 JOHN WHATLEY, M.D.

1849 JOHN WHITE.

1852 JOHN WIBLIN, M.D., Medical Inspector of Emigrants and Recruits; Southampton, *Trans.* 1.

1844 FREDERIC WILDBORE, 1, Trafalgar place east, [245] Hackney road.

1837 GEORGE AUGUSTUS FREDERICK WILKS, M.D.

1863 SAMUEL WILKS, M.D., Assistant-Physician to, and Lecturer on Pathology at, Guy's Hospital; 11, St. Thomas's street, Southwark.

1864 EDMUND SPARSHALL WILLETT, M.D., Resident Physician, Wyke House, Isleworth, Middlesex, and 7, Suffolk place, Pall Mall.

1860 ARTHUR WYNN WILLIAMS, M.D., 20, King street, Portman square.

1840 CHARLES JAMES BLASIUS WILLIAMS, M.D., F.R.S., Consulting Physician to the Hospital for Consumption; 49, Upper Brook street, Grosvenor square. C. 1849-50. V.P. 1860-1.

1859 *CHARLES WILLIAMS, House-Surgeon to the Norfolk and Norwich Hospital; Norwich.

1859 JOSEPH WILLIAMS, M.D., 8, Tavistock square.

1829 ROBERT WILLIS, M.D., Barnes, Surrey. L. 1838-41.

1839 †ERASMUS WILSON, F.R.S., Consulting Surgeon to the St. Pancras Infirmary; 17, Henrietta street, Cavendish square. *Trans.* 2.

1863 ROBERT JAMES WILSON, L.R.C.P. Edinb., 24, Grand Parade, St. Leonards-on-Sea, Sussex.

1850 *ROBERT STANTON WISE, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.

1825 THOMAS ALEXANDER WISE, M.D., Rostellan Castle, Rostellan, County Cork.

1841 GEORGE LEIGHTON WOOD, Surgeon to the Bath General Hospital; 27, Queen square, Bath.

Elected

1851 JOHN WOOD, Assistant-Surgeon to King's College Hospital, and Demonstrator of Anatomy in King's College, London; 4, Montague street, Russell square. *Trans.* 2.

1848 WILLIAM WOOD, M.D., Physician to St. Luke's Hospital for Lunatics; 54, Upper Harley street.

1843 JOHN WARD WOODFALL, M.D., Physician to the West Kent Infirmary; Maidstone, Kent.

1833 †THOMAS WORMALD, Surgeon to St Bartholomew's Hospital, 42, Bedford Row. C. 1839. V.P. 1854.

1842 WILLIAM COLLINS WORTHINGTON, Senior Surgeon to the Lowestoft Infirmary; Lowestoft, Suffolk. *Trans.* 3.

1848 EDWARD JOHN WRIGHT, 13, Montague place, Clapham road.

1855 HENRY G. WRIGHT, M.D., Physician to the Samaritan Free Hospital for Women and Children: 23, Somerset street, Portman square.

1860 JOHN WYATT, Surgeon-Major, Coldstream Guards; Hospital, Vincent square, Westminster.

[It is particularly requested, that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1841 WILLIAM THOMAS BRANDE, D.C.L., F.R.S., Hon. Professor of Chemistry at the Royal Institution of Great Britain, Member of the Senate of the University of London; Royal Mint, Tower hill, and 15, Calverley park, Tunbridge Wells.

1835 SIR DAVID BREWSTER, K.H., D.C.L., LL.D., F.R.S., Corresp. Memb. Institute of France, Principal and Vice-Chancellor of the University of Edinburgh.

1853 SIR BENJAMIN COLLINS BRODIE, Bart., M.A., F.R.S., Aldrichian Professor of Chemistry in the University of Oxford; Cowley House, Oxford.

1847 EDWIN CHADWICK, late Commissioner of the Board of Health.

1862 CHARLES GILES BRIDLE DAUBENY, LL.D., M.D., F.R.S., Hon. M.R.I.A., &c., Professor of Botany, Oxford.

1835 MICHAEL FARADAY, D.C.L., F.R.S., Corresp. Memb. Institute of France, Member of the Senate of the University of London, and Fullerian Professor of Chemistry in the Royal Institution.

1857 WILLIAM FARR, M.D., D.C.L., F.R.S., General Register Office, Somerset House, and Southlands, Bromley, Kent.

1841 SIR JOHN FREDERICK WILLIAM HERSCHEL, Bart., D.C.L., F.R.S., Corresp. Memb. Institute of France; Collingwood, near Hawkhurst, Kent.

1835 SIR WILLIAM JACKSON HOOKER, K.H., D.C.L., LL.D., F.R.S., F.L.S., Director of the Royal Botanic Gardens, Kew; West Park, Kew.

1847 RICHARD OWEN, D.C.L., LL.D., F.R.S., Corresp. Memb. Institute of France (Foreign Associate of the Academy of Sciences); Superintendent of the Natural History Departments in the British Museum; Sheen Lodge, Mortlake.

1825 The Rev. ADAM SEDGWICK, A.M., D.C.L., F.R.S., Woodwardian Professor of Geology, Cambridge.

1841 The Rev. WILLIAM WHEWELL, D.D., F.R.S., Master of Trinity College, Cambridge.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1841 G. ANDRAL, M.D., Member of the Institute and of the Imperial Academy of Medicine, Physician in Ordinary to the Emperor of the French, Professor of Pathology in the Faculty of Medicine; Paris.

1862 JEAN CRUVEILHIER, M.D., Physician to the "Hôpital de la Charité," Professor of Pathological Anatomy to the Faculty of Medicine, Member of the Imperial Academy of Medicine, &c.; Paris.

1856 BARON PAUL DUBOIS, Commander of the Legion of Honour, Member of the Imperial Academy of Medicine, Dean of the Faculty of Medicine; Paris.

1835 CARL JOHAN EKSTRÖMER, M.D., C.M., K.P.S., and W., Physician to the King of Sweden, President of the College of Health, and Director-General of Hospitals; Stockholm.

1841 CHRISTIAN GOTTFRIED EHRENBERG, Member of the Institute of France; Berlin.

1859 J. HENLE, M.D., Professor of Anatomy at Göttingen.

1841 JAMES JACKSON, M.D., LL.D., Emeritus Professor of Medicine in the Harvard University, Boston, U.S.

1856 BERNHARD LANGENBECK, M.D., Professor of Surgery in the University of Berlin.

1843 BARON JUSTUS von LIEBIG, M.D., Foreign Associate of the Academy of Sciences, Conservator of the Royal Collection, and Professor of Chemistry in the University of Munich.

1841 P. C. A. LOUIS, M.D., Honorary Physician to the Hôtel-Dieu, Member of the Imperial Academy of Medicine; Paris.

1847 CARLO MATTEUCCI, Professor in the University of Pisa, Member of the Institute of France; Minister of Public Instruction in Italy.

Elected

1853 VALENTINE MOTT, M.D., LL.D., Emeritus Professor of Surgery and Surgical Anatomy, in the University of New York; New York.

1841 BARTOLOMEO PANIZZA, M.D.; Pavia.

1862 NIKOLAUS PIROGOFF, M.D., Professor of Surgery to the Medico-Chirurgical Academy in St. Petersburg, Director of the Anatomical Institute, Consulting Physician to the Hospitals Obuchow, Peter-Paul, and Maria Magdalena; St. Petersburg.

1859 PIERRE RAYER, M.D., Grand Officer of the Legion of Honour, Member of the Institute, and of the Imperial Academy of Medicine; Paris.

1850 CARL ROKITANSKY, M.D., Curator of the Imperial Pathological Museum, and Professor of the University of Vienna. Referee for Medical and University Education to the Austrian Ministry.

1856 LOUIS STROMEYER, M.D., Director-General of the Medical Department of the Army of Hanover; Hanover.

1856 A. VELPEAU, Member of the Institute, and of the Imperial Academy of Medicine, Professor in the Faculty of Medicine, Surgeon to the "Hôpital de la Charité," President of the Academy of Sciences; Paris.

1856 RUDOLPH VIRCHOW, M.D., Professor of Pathological Anatomy in the University of Berlin.

CONTENTS.

PAGE

List of Officers and Council v

List of Referees vii

List of Presidents of the Society viii

List of Fellows of the Society ix

I. Case of Cancerous Infiltration of the Penis; with Cancerous Ulcer of the Bladder, and Secondary Deposits in the Lungs, Bones, and other parts of the Body. By HOLMES COOTE, F.R.C.S., Surgeon to St. Bartholomew's Hospital 1

II. Case of a Mucous Cyst on the Laryngeal Aspect of the Epiglottis, successfully treated by incision. By ARTHUR E. DURHAM, F.R.C.S, Assistant-Surgeon to Guy's Hospital. (Communicated by John Birkett, Hon. Sec.) . . 7

III. Case of unusual difficulty in Lithotomy, arising from great Distortion of the Pelvis by Rickets. By HENRY THOMPSON, F.R.C.S., Surgeon to University College Hospital; with a note by John Erichsen, F.R.C.S. . . 11

IV. Account of some unusual occurrences during the cure of a Popliteal Aneurism. By CHARLES H. MOORE, F.R.C.S., Surgeon to the Middlesex Hospital . . . 17

V. Case of Popliteal Aneurism successfully treated by Flexion of the Knee. By ARTHUR E. DURHAM, F.R.C.S., Assistant-Surgeon to Guy's Hospital 25

VI. Some Particulars of a Case of Popliteal Aneurism cured by Flexion of the Knee. By the late H. C. JOHNSON, F.R.C.S., narrated by ERNEST HART, F.R.C.S., Ophthalmic Surgeon to St. Mary's Hospital. (Communicated by John Birkett, Hon. Sec.) 29

VII. Note on the application of Indices to Aneurismal Clamps, and other pressure instruments. By ERNEST HART, F.R.C.S., Ophthalmic Surgeon to St. Mary's Hospital. (Communicated by John Birkett, Hon. Sec.) . . . 31

PAGE

VIII. Clinical Observations illustrating the effects produced by the implication of Branches of the Pneumogastric Nerve in Aneurismal Tumours. By S. O. Habershon, M.D., Senior Assistant-Physician to Guy's Hospital . . 35

IX. On the Endemic Hæmaturia of the Cape of Good Hope. By John Harley, M.D., Assistant-Physician to King's College Hospital. (Communicated by Dr. Lionel S. Beale.) 55

X. Some account of the Amputations performed at St. Bartholomew's Hospital, 1853-63. By George William Callender, F.R.C.S., Assistant-Surgeon to St. Bartholomew's Hospital 75

XI. A Case of Strangulated Femoral Rupture, where, on a former occasion, the neck had been torn from the body of the sac, in the taxis, and the escaping bowel had formed a subperitoneal pouch which finally attained an extraordinary size. By J. W. Hulke, F.R.C.S., Assistant-Surgeon to the Middlesex and Royal London Ophthalmic Hospitals . 97

XII. On the Absorption of Dead Bone. By William Scovell Savory, F.R.S., Assistant-Surgeon to St. Bartholomew's Hospital 103

XIII. On a new operation for obtaining union of Ununited Fracture, with remarks on its application to certain cases of recent fracture. By E. R. Bickersteth, F.R.C.S., Surgeon to the Liverpool Royal Infirmary. (Communicated by Dr. Murchison.) 113

XIV. On a new method of procuring the consolidation of Fibrin in certain incurable Aneurisms. By C. H. Moore, F.R.C.S., Surgeon to the Middlesex Hospital. With the Report of a case in which an Aneurism of the ascending Aorta was treated by the insertion of Wire. By Charles Murchison, M.D., Physician to the London Fever Hospital 129

XV. Two Cases of Stone in the Bladder of the Female, treated by rapid urethral dilatation; with remarks on the operation. By Thomas Bryant, F.R.C.S, Assistant-Surgeon to Guy's Hospital 151

XVI. Statistics of Queen Charlotte's Lying-in Hospital. By George B. Brodie, M.D., one of the medical officers for the out-patients. (Communicated by Charles Hawkins, Consulting-Surgeon to Queen Charlotte's Hospital.) . 169

XVII. Account of a Case of Aneurism of the Abdominal Aorta, which was cured by compression of that artery immediately above the tumour. By William Murray, M.D., Physician to the Dispensary, Newcastle-on-Tyne . . 187

XVIII. Case of Congenital Imperfection of the Mammæ, Sexual Organs, Sternum, and Heart, in a woman aged twenty-two years. By Edward Headlam Greenhow, M.D., Assistant-Physician to the Middlesex Hospital . . 195

PAGE

XIX. Pathological Researches into the Diseases of the Ear, Supplement to Seventh Series: Sebaceous Tumours in the External Auditory Meatus. By JOSEPH TOYNBEE, F.R.S., Aural Surgeon to St. Mary's Hospital, &c. . . 203

XX. On the condition of the Stomach and Intestines in Scarlatina. By SAMUEL FENWICK, M.D., late Lecturer on Pathological Anatomy at the Newcastle-upon-Tyne College of Medicine 209

XXI. On the origin, structure, and mode of development of the Cystic Tumours of the Ovary. By WILSON FOX, M.D. Lond., Professor of Pathological Anatomy at University College, London, and Assistant-Physician to University College Hospital 227

XXII. A successful case of Paracentesis Capitis. By THOMAS YOUNG THOMPSON, M.R.C.S., Newcastle-on-Tyne. (Communicated by Dr. Fuller, Hon. Sec.) . . 289

XXIII. On the Causes of Hernia. By JOHN A. KINGDON, F.R.C.S., Surgeon to the City of London Truss Society . . 295

XXIV. Report of the Scientific Committee on the Uses and the physiological, therapeutical, and toxical Effects of Chloroform, as well as into the best mode of administering it, and of obviating any ill consequences resulting from its administration 323

INDEX 443

LIST OF THE PLATES.

		PAGE
I.	Distortion of the Pelvis, in a Case of unusual difficulty in Lithotomy (Mr. H. Thompson)	16
II.	Endemic Hæmaturia of the Cape of Good Hope: microscopic diagrams of the Parasite and its ova (Dr. John Harley)	74
II*a*.	Ditto ditto (ditto) .	74
III.	Badly united Fracture of the Inferior Maxilla: Case of Samuel Smith before operation (Mr. Bickersteth) .	128
IV.	Ditto ditto, after operation (ditto) .	128
V.	Mucous Membrane of the Stomach in Scarlatina (Dr. Fenwick)	226
VI.	Surface of Small Intestine in Scarlatina; Mucous Membrane of the Stomach in Measles (ditto) . .	226
VII.	Tube-casts and Cells from the Stomach in Scarlatina; Hæmorrhage into a Sweat-gland in Scarlatina (ditto) .	226
VIII.	Cystic Tumours of the Ovary: figures 1 to 15 (Dr. Wilson Fox)	288
IX.	Ditto ditto figures 16 to 29 (ditto) .	288
X.	Ditto ditto figures 30 to 42 (ditto) .	288

Woodcuts.

Aneurismal Clamp with Indices (Mr. E. Hart) . . . 34

Aneurismal Tumour pressing upon the Pneumogastric Nerve, &c. (Dr. Habershon) 38

Ditto ditto (ditto) 45

Strangulated Femoral Rupture: side view of the Hernial Pouch (Mr. Hulke) 101

Ditto: front view (ditto) 101

Union of Ununited Fracture: left humerus (Mr. Bickersteth) . 123

Ditto: left humerus, compound fracture (ditto) . 125

Ditto: left tibia . . . . (ditto) . 126

Congenital imperfection of Mammæ, &c. (Dr. Greenhow) . . 197

A CASE

OF

CANCEROUS INFILTRATION OF THE PENIS;

WITH

CANCEROUS ULCER OF THE BLADDER, AND SECONDARY DEPOSITS IN THE LUNGS, BONES, AND OTHER PARTS OF THE BODY.

BY

HOLMES COOTE, F.R.C.S.,

SURGEON TO, AND LECTURER ON SURGERY AT, ST. BARTHOLOMEW'S HOSPITAL.

Received Oct. 21st.—Read Nov. 10th, 1863.

Cancer of the penis usually attacks the anterior part of the organ, the prepuce being in the contracted state called phymosis. It may assume the characters of epithelioma, or appear as a hard knot, which has been, in some instances, mistaken for a chancre. But general cancerous infiltration of the penis is, according to my experience, so rare, that I venture to bring the following case before the attention of the Society.

Richard C—, æt. 55, was admitted into St. Bartholomew's Hospital, September 10th, 1863, under my care, on account of enlargement, induration, and distortion of the entire penis, attended with severe pain. He had also a small nodular swelling on the right tibia.

He said that, although he had suffered slightly from

venereal disease in his youth, he had never, for very many years, experienced any trouble with his urinary organs until about eighteen months ago, when micturition became frequent, difficult, and painful. He applied for relief at another hospital, where catheters were daily passed. In consequence of this treatment much bleeding ensued. The swelling on the tibia was of eight months' duration.

The patient was sallow and emaciated. He seemed completely prostrate both in body and mind. There was complete phymosis; and from under the prepuce there flowed on pressure a sero-purulent, fetid, and offensive discharge. The penis was rigid, and of incompressible hardness. It was bent on itself, the concavity directed upwards, so that micturition was difficult in the extreme, and could only be accomplished by the patient throwing himself into strange and uncomfortable attitudes. He suffered so much pain at night that the repeated administration of morphia was necessary. On two occasions morphia was injected under the skin of the right arm, but the immediate effect was, contrary to our expectations and experience, such intense smarting pain, that this method of administering the remedy was abandoned, although its ultimate effect was satisfactory.

On September 12th his sufferings had been so great that he was removed to the operating theatre, with the view of obtaining a more complete examination of the parts while he was under the influence of chloroform, and of removing the entire penis, if by such a measure the whole of the morbid structures of that region could be taken away. But the chloroform, administered with every care, excited such an alarming degree of spasm of the throat, dyspnœa, and sinking of the pulse, that it was deemed imprudent to persist. The patient was accordingly replaced in bed.

Subsequently the prepuce was slit up with much relief. The glans penis was exposed, nodulated, hardened, and superficially ulcerated. The symptoms of general suffering, however, became more severe, and the patient expired on

October 16th, having become suddenly drowsy and comatose twenty-four hours before death.

Examination of the body.—Emaciation extreme; the whole surface of the integument was of a pallid, yellowish hue. The skin of the penis was soft, normal, and quite moveable. The corpora cavernosa were distended to their uttermost by the infiltration of a semifluid creamy deposit; this fluid exuded from the cut surface as a thick juice, and under the microscope was seen to contain nucleated cells, mostly elongated and caudate, very similar to those deposited in scirrhous cancer of the breast. There were also oil-globules and exudation corpuscles. The corpus spongiosum and the glans penis were infiltrated and hardened by the deposit of similar material. On closer and more minute inspection we found that the cancer juice was deposited within the venous sinuses of the penis, but cells were mingled with the fibrous tissue of the trabeculæ. The greater deposit was in the former situation. The urethra, compressed throughout its course, was smooth and normal within. Around the bulb and the prostate gland the tissues were matted together by similar deposit of cancerous matter.

The mucous membrane of the bladder was dark-coloured, sloughy-looking, and fetid. A large circular ulcer, with ragged, elevated, and indurated edges, occupied its surface at the lower and right side of the organ; patches of fungous growth projected from it in various situations. It measured two inches by one and a half, and involved the opening of the right ureter. The posterior wall of the bladder was broken down as if by an abscess, and the urine had been only prevented from flowing into the cavity of the abdomen by a number of circumscribed adhesions. The cavity of the bladder contained offensive puriform fluid.

Between the bladder and the rectum was a chain of glands filled with white, cancerous matter. Some few of the lumbar and iliac absorbent glands were similarly infiltrated. But the greater number were normal.

The right ureter was dilated and thickened, and traced

into the ulceration in the walls of the bladder, where it was lost. The right kidney was reduced to a small sac formed by the dilated pelvis and a small investment of secreting tissue. It contained no fluid, and did not even smell urinous. The left kidney was enlarged, weighed seven and a half ounces. It was pale, but apparently sound, and smelt strongly of urine when first cut open.

The heart was small, pale, and flabby; its fibres were in a state of extreme fatty degeneration, and this change was confirmed microscopically, as specially existing in the left ventricle.

The bronchial glands were full of soft, white, cancerous matter.

The lungs were generally emphysematous, and scarred here and there by old tubercular cicatrices. The opposed surfaces of the pleuræ were in parts united by old adhesions.

The pleural surface of the lungs was mottled here and there by white circumscribed patches. Cancerous deposits were scattered in larger and smaller masses throughout the organs, each deposit being surrounded by condensed pulmonary tissue.

The tumour on the shin-bone was composed of the same white, cancerous matter. It had been deposited under the periosteum, and the surface of bone on which it lay was roughened and discoloured.

Similar distinct collections of this matter were found springing from the tarsal bones of the right foot, and occupying the tendinous sheaths of the peronei muscles in the same limb.

This case is remarkable, in the first place, for the character and the extent of the cancerous deposit. Infiltrated true schirrus is rarely seen elsewhere than in the mammary gland; primary cancer in other parts is mostly of softer consistence, and is often a distinct outgrowth, as in epithelioma.

The extent of the primary deposit, occupying the entire

organ, is likewise singular. There is one preparation in the museum of St. Bartholomew's Hospital, described as tuberculous deposit, which bears so close a resemblance to the recent specimen before the Society, that I think it must be incorrectly registered. It has, however, been immersed in spirit so many years that it has lost those characteristics which would be available to microscopic examination. With this exception I have never met with a similar case. In the next place it is worthy of note how very slightly the absorbent system was affected in comparison with the very great extent of the disease in organs important to life. I believe we should have found cancerous deposit within the cranium had we been permitted to investigate that point at the post-mortem examination.

Lastly, the condition of the heart affords us a useful lesson to be careful in the administration of chloroform. Had the employment of the anæsthetic been continued, sudden death would in all probability have ensued. It was my great misfortune on one occasion to administer chloroform to a lady, who suddenly expired after a few seconds' inhalation. In this case, after death, we found fatty degeneration of the muscular tissues of the heart, produced by causes which were unknown to me before seeing the patient, and which had been intentionally concealed.

CASE

OF A

MUCOUS CYST ON THE LARYNGEAL ASPECT OF THE EPIGLOTTIS;

SUCCESSFULLY TREATED BY INCISION.

BY

ARTHUR E. DURHAM, F.R.C.S.,

ASSISTANT-SURGEON TO, AND LECTURER ON ANATOMY AT, GUY'S HOSPITAL.

COMMUNICATED BY

JOHN BIRKETT, HON. SEC.

Received Oct. 31st.—Read Nov. 10th, 1863.

THE following case is, as far as I have been able to ascertain, unique; and I trust it may be thought sufficiently interesting to merit for a few minutes the attention of the Fellows of the Royal Medical and Chirurgical Society.

J— H—, a delicate but very intelligent boy, æt. 11, was admitted into Clinical Ward, Guy's Hospital, under the care of Dr. Wilks, on the 10th of June, 1863. Two years previously he had suffered from a severe attack of sore-throat, and ever since had experienced more or less difficulty in swallowing. This difficulty had become very much greater during the last three months; during the same period his voice had gradually altered, becoming first more and more hoarse, and afterwards more and more feeble; he had also suffered from repeated attacks of severe dyspnœa, which came on most frequently, but not always, during sleep.

On admission, he complained of considerable pain, increased by pressure, about the larynx; he could only speak in a low, hoarse whisper; he had a dull, croupy cough; he could only swallow fluids with difficulty, and solids seemed, he said, to stick in his throat.

Three or four nights after admission he had so severe an attack of dyspnœa that tracheotomy was upon the point of being performed; but the operation was delayed, at the express desire of Dr. Wilks, until a thorough laryngoscopical examination could be made; and the patient was carefully watched during the night.

The following morning (15th) I was requested by Dr. Wilks to examine the patient with him, by means of the laryngoscope. I did so, and found that the epiglottis was not to be seen in its ordinary form; but instead of it there appeared a large round swelling, projecting downwards and backwards, and completely covering in and concealing the glottis. On either side of the posterior part of this tumour the aryteno-epiglottidean folds could be seen—pale, swollen, and apparently œdematous. The tumour could be just reached by the finger: it was pale, somewhat translucent, and on digital examination very tense. Feeling certain that it contained fluid, I proposed to make a free incision into it, and (Dr. Wilks fully assenting) I proceeded at once to do so by means of a long, curved, sharp-pointed bistoury, defended to within three-quarters of an inch of the point by sticking-plaister. My incision was followed by a sudden gush of thick, glairy, muco-purulent matter, mixed with a little blood. Momentary dyspnœa, arising from spasm of the glottis, followed; but this very speedily subsided, and all the symptoms from which the patient had so severely suffered were at once relieved: he could breathe better, speak better, swallow better; and in the evening he was singing in his bed.

The matter discharged from the tumour (which was evidently a mucous cyst) resembled exactly the contents of a ranula beginning to suppurate. It consisted of thick, glairy mucus, with epithelial-cells and a few pus corpuscles.

The presence of the pus showed the existence of inflammation; and this condition was also indicated by the œdema of the surrounding parts, the pain, and the general aggravation of symptoms.

Day by day, after the operation, the patient improved in health, and the parts recovered their normal condition. I made repeated laryngoscopical examinations, and was interested to watch the subsidence of the œdema in the surrounding parts, and the filling up and healing of the wound I had made.

The boy remained in the hospital, on another account altogether, until the middle of August, when he left quite well in every respect. I saw and examined him on the 3rd of October. He was perfectly well, but I succeeded in discovering a trace of the cicatrix of my incision on the lower part of the posterior (*i. e.* laryngeal) surface of the epiglottis.

CASE

OF

UNUSUAL DIFFICULTY IN LITHOTOMY,

ARISING FROM

GREAT DISTORTION OF THE PELVIS BY RICKETS.

BY

HENRY THOMPSON, F.R.C.S.,

SURGEON TO UNIVERSITY COLLEGE HOSPITAL;

WITH A NOTE BY

JOHN ERICHSEN, Esq.,

PROFESSOR OF SURGERY IN UNIVERSITY COLLEGE, AND SURGEON TO THE HOSPITAL.

Received Nov. 17th.—Read Nov. 24th, 1863.

I HAVE recently met with a form of obstruction to the removal of stone from the bladder, which is of so rare a kind that it appears to me desirable to place it on record. There are few professional duties, perhaps, which more urgently demand performance than that of citing those irregular and remarkable cases which form exceptions to our common experience; more especially if the knowledge of them may be useful to guide others when encountering similar instances hereafter. In the present case I am the more impelled to do so, because I know of no parallel example in the literature of the subject. Appended to the recital which follows are some remarks by my friend and

colleague, Mr. Erichsen, under whose care the patient had previously been placed.

Case.—George Sharp, æt. 4½. I saw him first August 20, 1863. He had the usual symptoms of stone in the bladder, while a cicatrix in the perineum indicated the site of a median operation of lithotomy which had been performed by Mr. Erichsen about nine or ten weeks before. There was a small fistulous opening in the cicatrix, through which some urine passed occasionally. The child was extremely weak and emaciated, and his sufferings were very severe.

On sounding I found a stone of rather large size, and I proposed to his parents to cut him at the earliest opportunity. There was evidently no alternative between this proceeding and a painful, lingering death. I was at this time in temporary charge of Mr. Erichsen's duties during his absence from town, and I obtained his full concurrence with my proposal before carrying it into effect.

The child was admitted into the hospital of University College on the last day of August, 1863.

On the 2nd of September he was placed on the operating table under chloroform, Mr. M. B. Hill holding the staff. I made my incision precisely in the line of the old cicatrix, and on my finger entering the neck of the bladder, I stated to those around that I found "an unusual prominence, which is evidently the promontory of the sacrum, deformed apparently by rickets, and barring the entrance of my finger into the cavity of the bladder, which viscus appears to be wholly in the abdomen." I felt a stone with the tip of my finger, which had just room to pass into the space between the promontory and the pubic symphysis. I endeavoured to pass a pair of small forceps on my finger, but the room was insufficient. On placing my right hand on the abdomen, I felt the stone most distinctly, and could place it by pressure in contact with the tip of my left index finger, situated in the neck of the bladder. I desired Mr. Hill to grasp the stone there, and to press it as much within the brim of the pelvis as possible. He maintained it in

that position firmly, while I insinuated a pair of polypus forceps, one blade laterally on either side, and after one or two fruitless attempts, from external layers of the calculus becoming detached, and causing the blades to slip, I succeeded in extracting it entire. There was not much bleeding; a tube was placed in the wound, and the child removed to bed.

The next day he showed symptoms of peritonitis, which increased, and on the third he died.

At the post-mortem examination the usual marks of peritonitis were found in the abdomen. The bladder was situated entirely in the abdominal cavity; the index finger could only just be passed flatwise between the sacral promontory and the symphysis pubis; the bladder was inflamed also. I had the pelvis carefully removed entire, and a drawing made of the preparation (see Plate I, Fig. 1*a*). Viewing the upper outlet of the pelvis, it is seen to present an irregular heart-shaped figure, obliquely cordate: the sacral promontory approaching within three-eighths of an inch of the left pubic ramus, and within barely five-eighths of the right. From the promontory to the symphysis pubes there is barely seven-eighths of an inch of space; and it must be remembered that these are the measurements on the dried bone, and that they were less when the soft parts were present. The calculus itself is also presented to the Society. It is composed of uric acid, is hard, and has an oval form with the following measurements, after losing some portions of its outer layer, which will be found accompanying it.

Length, one inch and an eighth; breadth, almost seven-eighths of an inch (that is, more than thirteen-sixteenths); thickness, fully five-eighths of an inch. It weighs two and three-quarter drachms. It is easily seen, by examining the preparation, that sufficient room to withdraw the stone through the pelvis exists only at the central portion of the outlet, and a little to the right of it. The length and the thickness are seen at Plate I, figs. *b* and *c*, which figures delineate the calculus of its exact form and size.

I have before said that I have not met with any record of a case presenting the difficulty here described. There is an allusion to it in Dr. Gross's work 'On the Urinary Organs,' 2nd Edit., p. 563, in the following terms:

"Embarrassment of a serious, if not insurmountable, character may arise from unusual narrowness of the outlet of the pelvis. In rickety subjects the opening is sometimes reduced to a mere vertical slit. Some years ago a soldier, affected with vesical calculus, died in one of the hospitals of Paris, and on examination it was found that the distance between the branches of the ischiatic and pubic bones did not exceed six lines."[1]

On reference, however, to the French journal named, I do not arrive at the conclusion given in the passage quoted. That a man so deformed died and was dissected, is clear; but there is no mention of his having been the subject of stone in the bladder.

The paragraph in the 'Journal des Progrés' is part of a long memoir 'On the Influence of Anatomical Deviations upon Surgical Operations,' by Dr. F. Robert; and it is as follows:

"The ischiatic and pubic rami of the pelvis may be more or less widely separated than usual. Sometimes we see them approximate so closely that they are only five or six lines distant from each other. I had the opportunity of observing such a condition in the body of a soldier at the Hospital of Val-de-Grace. This configuration involves the impossibility of executing perineal lithotomy with success. It is one of those cases in which supra-pubic lithotomy must be resorted to."[2]

[1] 'Journal des Progrés,' t. viii, p. 200.

[2] The original of the quotation, translated from the French. "Les branches ischio-pubiennes de l'os coxal sont plus ou moins ecartées. On les a vues tellement rapprochées l'une de l'autre qu'elles n'etaient distantes que de 5 or 6 lignes. J'ai l'occasion d'observer un fait analogue sur le cadavre d'un soldat à l'hôpital du Val-de-Grace. Cette configuration entrâine avec elle l'impossibilité d'executer la taille sous-pubienne avec succés. C'est un des cas où il faudrait recourir à l'incision de la

It is quite obvious that the reference to perineal lithotomy is a general reflexion suggested by the abnormal condition of the pelvis, and is not the history of any case in which an operation had been performed. Hence I am unable to adduce an instance illustrating the one under consideration. There exists consequently no experience to guide us as to the propriety of adopting what, at first sight, appears to be the preferable mode of proceeding, viz., that of suprapubic lithotomy. Had I been at all aware of the anatomical conformation of the pelvis in this case before operating, the question of adopting that procedure would have been seriously considered by me. It is not unlikely, however, that the purely abdominal situation of the bladder might have rendered that operation scarcely less hazardous than the perineal one. I think, however, that I should, with my present knowledge, elect to adopt it in a precisely similar case.

With regard to the mode of death which terminated the patient's existence, it was that which, after considerable research on this subject, I have elsewhere shown to be the most common one among the fatal cases of children, viz., peritonitis.[1] The natural situation of the bladder in childhood renders it as much an abdominal as a pelvic organ, and difficult lithotomy, or the access of inflammation from any other cause, generally attacks the peritoneal more readily than the pelvic connections of the viscus. In the case before us this was the course observed, and although extraction was not particularly difficult nor very prolonged (about seven or eight minutes), yet in the bad condition of the patient the occurrence of peritoneal inflammation, under the circumstances described, could not occasion much surprise.

vessie au-dessus du pubis."—"De l'influence des Variétés Anatomiques sur les Opérations Chirurgicales," par F. Robert. 'Journal des Progrés des Sciences et Institutions Médicales,' Paris, 1828, tome viii, p. 200.

[1] 'Practical Lithotomy and Lithotrity,' by Mr. H. Thompson, p. 95, Lond., 1863.

Note by Mr. Erichsen.

The patient whose case has been described by Mr. H. Thompson was, in the first instance, admitted under my care into University College Hospital for stone in the bladder. He was an unhealthy, rickety child, with considerable tumefaction of the abdomen and distortion of the lower limbs.

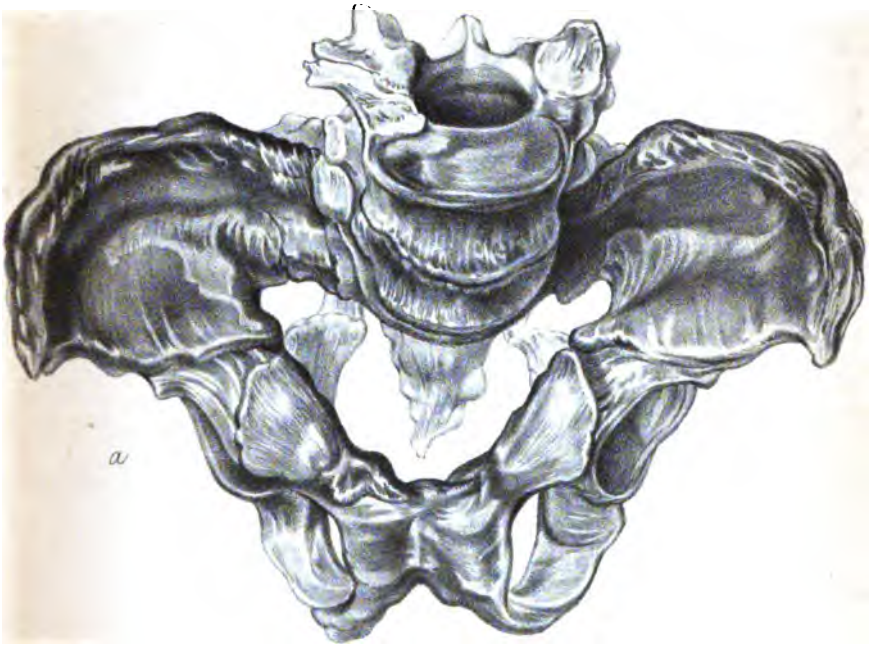
I cut him for stone on June 10th, by the median operation, as now usually practised. After dilating the neck of the bladder, the finger passed into a narrow tubular cavity, at the upper end of which the stone could be felt by me high above the pubes. I readily seized it with ordinary lithotomy forceps, but found, on endeavouring to extract it, that it was either dragged out from between their blades, or that the calculus and forceps got locked together in the narrow channel leading to the external wound. It was evident, at the time of the operation, that the obstacle to extraction did not exist in the soft parts, and I consequently did not attempt to enlarge the aperture in them by incision or by forcible dilatation. Finding that the calculus could not be extracted in the usual way by ordinary instruments, and that it was too hard to be broken up by the forceps and extracted piecemeal, I thought it more prudent to desist from further attempts, and to endeavour to complete the operation at some future period. The child was accordingly sent back to the ward, where it remained about a month, the wound healing kindly up to a fistulous aperture. It was then attacked by diarrhœa, which necessitated its removal from the hospital, the mother being directed to bring it back for operation when, sufficiently recovered. It was readmitted towards the end of August, when, during my absence, it passed into the hands of Mr. Thompson, who performed the operation he has just described.

DESCRIPTION OF PLATE I.

Fig. *a*.—The pelvis, from Mr. Thompson's case of distortion occurring in a child, the subject of stone in the bladder. The outlet is of the natural size.

Fig. *b*.—The calculus. Actual size.

Fig. *c*.—Ditto, to show the thickness.



William Bagg del et lith.

W. West. imp.

AN ACCOUNT OF SOME UNUSUAL OCCURRENCES

DURING THE CURE OF A

POPLITEAL ANEURISM.

BY

CHARLES H. MOORE, F.R.C.S.,

SURGEON TO THE MIDDLESEX HOSPITAL.

Received Nov. 19th.—Read Dec. 8th, 1863.

THE recent employment of the iodide of potassium in the treatment of aneurism induces me to make known the following case, in which I employed it for a short time. I am further led to offer the case to the attention of the Fellows, because of some unusual occurrences in the progress of it, which I happened to possess peculiarly favorable opportunities of studying.

The aneurism was in the calf, under cover of the highest part of the gastrocnemius. Four weeks after it had been first observed it occupied the entire breadth of the limb, and was as large as the fist. It approached the surface in three places—on either side of the gastrocnemius, and between the heads of that muscle in the lower part of the popliteal space. Its outer projection was much the largest, and the most strongly pulsating, and it extended six inches vertically behind the fibula. The beat was forcible and expanding, so as to be visible through the trousers, and the bruit was distinct and characteristic. About one third of the blood left the sac when the circulation in the limb was arrested. There was venous conges-

tion, with œdema and neuralgic pain in the lower part of the limb. The anterior tibial beat feebly, the posterior not at all. Pulse 100.

The patient was a well-nourished, athletic man, of very active habits, and fond of field sports. Though only forty years of age, he had a wide gray circle around either cornea; and my friend, Dr. Greenhow, who had recommended him to my care, detected a rough systolic bruit in the ascending aorta. The aneurism was spontaneous in its origin, and first attracted notice by the intense pain produced in it on crossing the legs, when it struck against the patella of the opposite limb.

From the form of the aneurism, and the greater force of the beat on its outer side, it appeared probable that the artery had given way at its external and posterior part. The extent of the expansion and recoil showed the opening in the artery to be a large one.

August 25th, 1863.—The treatment by genuflexion was first employed, and was continued for eight days. The pulse came down to about 80, and the force of the beat of the aneurism diminished. Though at first tolerable, the bending of the knee brought on a pain along the posterior tibial nerve, which increased until it could not be endured. The angle of flexion was enlarged from time to time, but it was found that any position of the knee which influenced the beat of the aneurism eventually produced insufferable torture in the sole of the foot. This method of treatment was accordingly abandoned.

September 1st.—I employed two tourniquets to make pressure on the femoral artery alternately in the groin and thigh. These instruments were taken off at night and twice during the day; but effective pressure, such as materially to lessen the pulsation of the aneurism, was made with them through ten or fourteen hours of each day. It was maintained until the 25th, with the exception of a few days, during which some soreness of the skin made it necessary to interrupt the compression in the groin. Between the 4th and the 21st the patient also took in-

ternally an ounce and a half of the iodide of potassium. During this period of twenty-five days considerable changes took place, which may be thus summarily recounted:

1. The pulse increased in frequency, sometimes reaching 120 in the minute, and never being less than 90.

2. The aneurism enlarged on the outer side, and also towards the popliteal space. These enlargements followed the temporary shortening of the daily compression, which was required by the tenderness of the skin, and they were accompanied by decided local pain in the parts of the aneurism which yielded to the distending force.

3. The aneurism felt much firmer; but this was to some extent a deceptive feeling. It was, indeed, firmer, inasmuch as it contained more coagulum, but it was also permanently tense; and this tension could not, by once feeling the tumour, be distinguished from hardness. Observing the changes, however, which took place from day to day, and from one part of a day to another, I was able to satisfy myself that the permanent tension was due to frequency of the pulse. At the rate of 120 in the minute the interval between the beats was too short to allow of more than a slight reflux of blood into the artery; the aneurism being full, beat little, and its wall was constantly distended and hard; with the pulse at 90 the aneurism expanded and contracted again, and some of the softness of fluid could be distinguished in its swell and its recoil. This distinction could be confirmed by arresting the circulation through the artery at the groin, when some estimate could be formed of the actual amount of solid matter in the sac. Moreover, even with the pulse at 120, the instrumental compression softened the beat of the aneurism, and lessened the deceptive firmness of its wall.

4. New solid substance appeared to be formed in the cavity of the aneurism.

5. Much soreness of the aneurism was felt, which the patient compared to that of a boil. It was very tender, so as not to bear pressure against a pillow, and was occasionally hot to the touch.

6. While the aneurism enlarged towards the ham, the pain in the foot diminished. At the same time a numbness of that part and inability to flex the toes coming on, showed that the posterior tibial nerve was not yet released from pressure. A new pain arose in the outside of the foot and little toe, as the aneurismal wall yielded externally towards the peroneal nerve.

Notwithstanding the enlargement, and other adverse symptoms thus detailed, the aneurism seemed, towards the end of September, to be approaching its cure. On the 25th there was great swelling of the saphena and cutaneous veins, and the blood could scarcely be pressed along them with the finger. The integuments were dark and congested, and there was increasing œdema. The anterior tibial was no longer to be felt on the instep, and the limb soon chilled on exposure. The beat of the aneurism was less than it had ever been. By the next day, the 26th, some collateral arteries had perceptibly enlarged, and the patient had a very distinct and obtrusive feeling of trickling about the limb. The skin of the leg had changed from a dusky to a bright pink hue, and its temperature was high, though still easily reduced. Half, at least, of the contents of the aneurism, when examined with the femoral artery compressed, appeared to be solid. There was thus a prospect of a speedy cure of the aneurism.

On the 27th this hope was disappointed. As the patient was seated at dinner, with his knee raised and bent, and without the tourniquets, a very severe, sharp pain suddenly struck him in the aneurism. It appeared to shoot to the middle of the calf, and was over in two or three minutes. He at once felt relieved of his former pain in the flexure of the knee. In a couple of minutes more he perceived that the sensibility had returned in the sole of the foot, and that in the same abrupt way the power of flexing the toes was regained. The feeling was still not quite perfect, and a tingling came on in all the parts of the foot which had just previously been benumbed. I examined the aneurism immediately, and observed that it had fallen in at its uppermost

part, the top of the calf no longer bulging, as it had done in the morning and during the previous days, into the femoral hollow of the ham. The pulsation also was lessened. The patient himself was conscious that some sudden change in the aneurism at the moment of pain had produced the alteration in his feelings, and had freed the back of his knee; and he continued throughout the evening intensely alive to the aneurism, and to what might be going on in it. He could hardly cease from examining the limb, and the occupation of his mind about it was peculiar and obvious. I concluded that a large piece of lymph had been detached from the uppermost part of the aneurism, and had released the popliteal nerve from pressure; that it was probably moving about in the currents of blood; and that, if fortunately carried into the arterial opening, it might finally plug it, and cure the aneurism.

No such event, however, took place, and on the next day the venous congestion had almost subsided, the œdema had lessened, and the trickling feeling of collateral circulation was scarcely perceptible. At a later period the anterior tibial artery was again felt beating feebly on the instep. The aneurismal impulse also became more soft and expansive; and it was clear that the remarkable change which had taken place in the disposition of the clots had released the popliteal artery and vein from compression, equally with the nerve. The circulation in the limb being thus restored to the natural channels, the newly enlarged collateral vessels had ceased to be necessary, and had again subsided.

In the night and the two days following the sudden change just described he occasionally suffered severe pain. Sometimes this was plainly in the aneurism itself, but on two occasions it was in the soleus muscle. An uneasiness came on at the inner part of that muscle, which in half a minute extended all over it, and speedily became a severe and unmistakeable cramp, causing him great distress for nearly an hour. The pains in the aneurism were relieved by compression of the femoral artery, but the cramp was relaxed only by stretching the muscle, by friction, and by

the application of heat. On the day following the last of these attacks of pain, September 30th, the aneurism was first distinctly felt as a limited sac, over which the loose and wasted muscles of the calf could be easily moved. From that time he had no more pain in the aneurism.

For the three following weeks the pressure by means of tourniquets was steadily kept up every day. At the advice of Dr. Greenhow, the patient took steel and hydrochloric acid in place of the iodide of potassium, and at dinner a little claret or port wine. His pulse declined to 86, and sometimes to 78, and his comfort was much increased under this treatment. The aneurism became more nearly globular in form, its lower and outer part falling in. The entire diminution of its size, however, was but slight, and the beat, though not widely expansive, was again soft.

Under these circumstances I thought the treatment by instrumental compression likely to be much prolonged, and on the 21st, in preparation for continuous digital compression, I left off all use of the tourniquets, and allowed the skin to recover itself. During this short interruption of the pressure on the artery the aneurism distinctly enlarged, and the foot became again uneasy.

October 22nd.—Digital compression was made for periods of two hours, alternately with pressure with a tourniquet for half an hour. This pressure was borne with little interruption for thirty-five hours, and was generally well maintained. Some of the great irksomeness attending it, and the pain it produced in the groin and back, were relieved by morphia; but at the end of the second day the patient was too exhausted and in too much pain to continue it, and it was interrupted for fourteen hours. It was noticed that the pressure required to control the artery was at first very moderate, but that after a few hours the vessel appeared to regain so powerful a beat that those assistants who came unfatigued to the work could only arrest its pulsations by exerting a considerable force. So simple a remedy as a cup of tea reduced the arterial impulse, and lessened by some pounds the pressure needful to arrest it.

On the following day, the 24th, the digital compression was renewed, and was well maintained for twelve hours. The artery sometimes, indeed, slipped from under the fingers; but on several occasions through the day no motion was felt in the aneurism for fifteen or twenty minutes, and it was rarely allowed to pulsate with its full force. The beat, when it did occur, was still soft. The exact moment of its cessation could not be ascertained; it was indicated by no pain or other feeling; and I was only able to observe that, during about twenty minutes of steady pressure, the tumour was firm and motionless. In a short time a collateral artery, on the inner side of the aneurism, began to beat with much force, and the feeling of trickling in the limb or of feathers being drawn along it returned. In three hours after the cessation of the beat in the aneurism the temperature of the leg had risen 5° of Fahrenheit above that of the healthy limb. A tourniquet was kept on the thigh, and moderate pressure made on the femoral artery throughout the night. The patient did not move from his position.

25th, 10 a.m.—At about this time, twelve hours and a half after the stoppage of the beat, a slight pulsation recommenced in the aneurism. It was deep, and not soft; but in half an hour it had increased, and became a little expansive. A feeble but unmistakeable systolic bruit had also returned.

Digital compression was accordingly renewed, and was thoroughly maintained for an hour and a half. By that time the pulsation and bruit had again ceased. At 12.35 p.m., half an hour after this event, the temperature of the leg had fallen to 84°; by 12.55 p.m. it was at 88°; at 1.30 p.m. it was 91°; at 3.20 it was 94°. The temperature of the right limb throughout this time did not vary much from 88°. In the evening the descending external circumflex was distinctly felt in the thigh, and the pulsation of the collateral arteries deceived most persons, on first feeling it, into the belief that the aneurism itself was still beating.

On the morning of the 26th the temperature of the aneurismal limb was 95°, and after this it varied, being on

the 27th 90°, and on the 28th 98°. By the 31st it was about equal to that of the healthy limb.

On the 6th of November the aneurism had diminished a little in size, and was everywhere rather soft; the leg and foot had lost much of their œdematous swelling. The anterior tibial was again perceived distinctly pulsating on the instep. There was no trace of the beat of the posterior tibial.

Postscript, April 26th, 1864.—The patient was seen this day, and found to be in good health. No definite tumour could be distinguished in the site of the aneurism; but the top of the left calf felt bulky and firm in contrast with the suppleness of the opposite limb. There was still a little œdema of the lower part of the leg, and slight numbness in parts of the foot. The anterior tibial beat well, and the foot had not felt cold throughout the winter. The limb had not been freely used in walking, in consequence of the relation of the tumour to the gastrocnemius. When the knee was extended the heel rose, and when the foot was laid flat on the ground the knee bent. This hindrance had, however, so much lessened, that the patient could stand and walk with the knee not far from straight.

CASE

OF

POPLITEAL ANEURISM,

SUCCESSFULLY TREATED BY FLEXION OF THE KNEE.

BY

ARTHUR E. DURHAM, F.R.C.S., &c.,

ASSISTANT-SURGEON TO AND LECTURER ON ANATOMY AT GUY'S HOSPITAL.

Received Nov. 21st.—Read Dec. 8th, 1863.

E. P—, a pale, unhealthy looking man, æt. 31, was admitted into Guy's Hospital, under my care, on the 5th August, 1863. He stated that he was by occupation a waiter, that his habits were temperate, but his general health was not good; he was often excitable, often languid, never very strong. Ten or twelve weeks before admission he experienced about the right knee, especially about the knee-cap, some slight pain, which after lasting for a few days subsided. In the course of two or three weeks, however, the pain returned with increased severity, and he discovered in the popliteal space a small pulsating swelling, which gradually increased in size until the period when he sought admission to the hospital. He suffered very severe pain whenever he attempted to bend the knee, and when walking, often became sick and faint. He could not recollect to have strained the limb, nor to have met with any accident to which he could attribute the origin of his malady.

On examination the day after admission, an aneurism,

about the size of an orange, was found situated rather in the upper part of the right popliteal space. It pulsated very distinctly, was comparatively firm to the touch, and could not easily be emptied to any extent by manipulation. The characteristic bruit could be clearly heard by means of the stethoscope. Pulsation in the aneurism was very readily stopped by pressure upon the femoral artery in the upper part of the thigh. Pulsation of the tibial arteries was distinct enough during extension, but could scarcely be felt when the limb was flexed. The patient was in a weak, irritable condition; his pulse was 96; he had evidently suffered much from pain and sleeplessness.

The case appearing in many respects suitable for the treatment by flexion, I resolved to try that method. Accordingly, the knee was bent as far as was deemed expedient, and the foot placed in a kind of slipper, which was attached by a heel-strap to broad bands passed round the thigh and pelvis. The limb was thus kept in position in a manner similar to that described by Mr. Spence in the 'Edinburgh Medical Journal' for November, 1858. At the same time a draught, composed of ten minims of tincture of opium, ten minims of tincture of digitalis, two minims of dilute hydrocyanic acid and camphor mixture, was ordered to be taken every three hours.

During the night the patient suffered so much pain and inconvenience that he was obliged to loosen the strap to the fullest possible extent, and in the morning his foot and leg were found to be somewhat œdematous. His pulse, however, was reduced from 96 to 75, and he felt generally more comfortable. Under these circumstances the slipper apparatus was discarded, and the plan recommended by Mr. Hart adopted. The limb was bandaged from the toes to within a short distance of the knee, and carefully flexed. A large pad of cotton wool was placed over the aneurism, and in the flexure of the joint, and the roller was then carried round the thigh. The thigh was, of course, flexed on the pelvis, and the whole limb supported by pillows. On each succeeding day for the next three days the flexion was

slightly increased, a fresh roller being placed over those already applied, which were not interfered with. On the 11th (*i.e.* the *fourth day from the fair commencement of the treatment*), all the bandages were removed, and the aneurism was carefully examined. No pulsation could be felt; but on making deep pressure into the space, a slight thrill was just perceptible. The limb was again flexed, and the bandages reapplied. The next day neither pulsation, thrill, nor bruit could be detected. During the *first two days* of the treatment the patient suffered *severe pain*; but during the last three he was comparatively easy. He was kept in bed for a few days, and directed gradually to extend the limb. In the course of a week or two he was able to get about comfortably. He suffered little or no pain, and the cured aneurism gradually diminished in size. He left the hospital quite well on the 28th August. Pulsation was distinct in the tibial arteries.

Postscript, July 26th, 1864.—The patient is again under my care in Guy's Hospital on account of a fistula in ano. No trace of the aneurismal sac can be felt, nor can any pulsation of the tibial arteries be discovered at the present time, though distinct enough when he left the hospital last August.

SOME PARTICULARS OF A CASE

OF

POPLITEAL ANEURISM,

CURED BY FLEXION OF THE KNEE.

BY THE LATE

H. C. JOHNSON, Esq.,

SURGEON TO ST. GEORGE'S HOSPITAL.

NARRATED BY

ERNEST HART,

OPHTHALMIC SURGEON TO ST. MARY'S HOSPITAL.

COMMUNICATED BY

J. BIRKETT, Hon. Sec.

Received Dec. 8th.—Read Dec. 8th, 1863.

My late much esteemed and lamented friend, Mr. H. C. Johnson, communicated to me more than a year since some details of a case of popliteal aneurism in which he had effected a cure by the flexion of the knee carried on in the manner which proved successful in the first case which I had the honour to communicate to this Society. The circumstances under which the cure was effected were somewhat peculiar, and Mr. Johnson had the intention of bringing forward a statement of the case. That intention having been unhappily frustrated, and as he had already authorised me to make use of the case, I thought it might be desirable briefly to record it as a pendent to that of Mr. Durham to-night.

The patient was an adult male, admitted into St. George's Hospital, under the care of Mr. H. C. Johnson, with a popliteal aneurism of moderate size, and of a few months' duration. Mr. Johnson employed pressure with tourniquets for nearly three months, but ineffectually, and was afterwards on the point of proceeding to ligature the femoral artery when flexion of the knee was suggested by the perusal of my case. He bandaged the leg to the thigh, including the whole foot beneath the bandage (as it is best, I think, to do), and the result was consolidation in six days. The cure was permanent. The previous failure of compression makes the case very interesting, and recalls that[1] in which Mr. Spence, of Edinburgh, found flexion successful in curing a relapsing popliteal aneurism which had recurred after ligature of the femoral at Scarpa's triangle, where the two unpromising alternatives were ligature of the iliac under peculiarly unfavorable conditions, or the old operation by cutting into the sac and tying the artery immediately above and below it, of which the mortality in this region has been very great.

The cases to-night recorded raise to twelve the number of cures of popliteal aneurism by the flexion method effected by British surgeons since September, 1858, the date of my first case originally brought before this Society.

Postscript, July, 1864.—Since the reading of this paper, a successful application of this method has been made by Mr. J. Hutchinson at the London Hospital.

[1] 'Edin. Med. Journal,' Nov., 1859, p. 434.

NOTE ON THE APPLICATION

OF

INDICES TO ANEURISMAL CLAMPS,

AND OTHER PRESSURE INSTRUMENTS.

BY

ERNEST HART,

OPHTHALMIC SURGEON TO ST. MARY'S HOSPITAL.

COMMUNICATED BY

J. BIRKETT, HON. SEC.

Received Dec. 8th.—Read Dec. 8th, 1863.

THE marked want of success which has attended the use of clamps and tourniquets in this country, as applied for the cure of aneurism, especially popliteal aneurism, by indirect pressure on the femoral, has become very evident to me in collecting published cases, and comparing the results in English hospitals with those which have been obtained by the Irish surgeons, who feel for this method of treatment all the interest which parentage can give, and are proportionately careful in applying the pressure. It is unnecessary to give figures or details for the purpose which I have in view, and I would only say that the analysis of unsuccessful cases shows that the greater part have failed through the pain caused by the pressure, through the ulceration or sloughing of the skin beneath the pad, or the extreme tediousness of the process causing its abandonment as ineffectual, and recourse to the ligature of the main artery.

Ligature, after compression has failed, gives less satisfactory results than primary ligature. In thirty-seven cases in which it was employed, after compression had failed, by English surgeons, and which I have collected, there were fourteen deaths.

The causes of a large number of the accidents of sloughing, ulceration, extreme pain, which I have mentioned, seem in a number of instances to be the consequences of defective application of the pressure.

To maintain continued and graduated pressure upon the femoral artery (even with the best compresses), without exerting too much pressure on the one hand, and yet sufficiently controlling the flow of blood on the other, is by no means easy, and requires skilled and constant attention, and a great amount of care is needed to prevent excess or defect. The most usual faults on the part of those who are entrusted by the hospital surgeon with watching these cases are employing an excess of pressure, or employing pressure in a wrong direction, so that power is wasted in compressing muscles, and the artery is not fixed against the bone.

The surgeon places the pad carefully in position, but it must presently be relaxed, and then the assistants commonly err, in more or less degree, in reapplying the pressure.

The application of indices to the instrument is likely, I think, to obviate much of the difficulty and to remove some prevalent causes of the failure of instrumental compression. The surgeon putting the pad in position can in each case ascertain for himself with what amount of pressure the femoral circulation can be stopped or slackened, accordingly as he desires to produce the one or other effect. Having determined the minimum force by which this can be effected, he can then inform his assistants, and point out to them the limits within which they must keep the pressure. If, in subsequently replacing the instrument, they find that they fail to control the circulation with that degree of pressure which has been experimentally ascertained to be sufficient, they will be aware that the pad is not well placed, and instead of screwing down the clamp and increasing the pres-

sure until the pulsation is stopped, they will be careful to improve the direction of the pressure. It is very useful for the surgeon himself to know what amount of pressure he is employing to stop the flow of blood; it is still more useful for the assistants to have under their eyes a constant indicator of the degree of correctness and care with which they are carrying out the surgeon's directions.

To give a long table of estimations of the degree of pressure required in different subjects would not, I think, serve any useful purpose, as this will need to be ascertained in each particular case at the commencement of the treatment. I may, however, just select the three following cases from my notes of the results of experiment on this point, as giving average figures for their typical subjects.

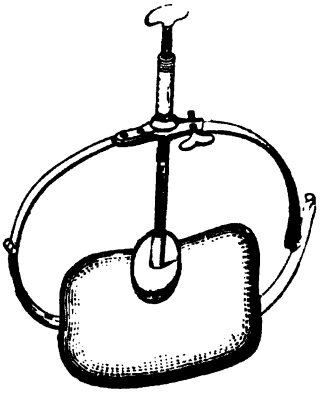
A. D—, adult male, 5 feet 7 inches high, somewhat emaciated after illness, measuring (at the level of the perinæum) around the thigh 15 inches, immediately above the knee 12 inches. Pulsation arrested at apex of Scarpa's triangle by pressure of 7 lbs. Pulsation arrested at apex of Hunter's canal by pressure of 8 lbs.

J. R—, adult male, 5 feet 6 inches high, robust and powerful, measuring (at level of perinæum) around thigh 22 inches, immediately above knee 14½ inches. Pulsation arrested at apex of Scarpa's triangle by pressure of 11 lbs. Pulsation arrested at Hunter's canal by pressure of 14 lbs.

A. N—, adult male, 5 feet 4 inches high, slight and feminine build, measuring (at level of perinæum) around thigh 20 inches, immediately above knee 12½ inches. Pulsation of femoral arrested at groin by pressure of 4 lbs. Pulsation of femoral arrested at Scarpa's triangle by pressure of 10 lbs. Pulsation of femoral arrested at Hunter's canal by pressure of 11 lbs.

In the instrument which I have made, and of which I append a woodcut, the pressure is registered by a needle on a scale. It is effected by a strong spring, which affords an

elastic pressure capable of nice graduation from four to twenty pounds. This instrument has already been tested in practice, and works well.



I owe to the kindness of the Director-General of the Army Medical Department the report of a case in which it answered expectation in curing a popliteal aneurism. The analysis of cases shows that elastic pressure is better tolerated than any other.

The principle involved in the application of indices seemed to me not unworthy the brief attention of this Society. It is obviously one which may be applied with advantage to other instruments of pressure and extension in surgery, and Messrs. Savigny and Co. have also constructed an index of similar kind applied to pulleys for reducing dislocations. It may be useful and instructive to apply them to apparatus for overcoming fibrous anchylosis of joints, restoring deformed limbs, and other similar surgical conditions.

CLINICAL OBSERVATIONS

ILLUSTRATING THE EFFECTS PRODUCED BY THE

IMPLICATION OF BRANCHES OF THE PNEUMOGASTRIC NERVE

IN

ANEURISMAL TUMOURS.

BY

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THE pneumogastric nerve is, perhaps, the most complicated nerve in the body, and the symptoms of disease by which it is implicated vary according to the part that is affected. As a sensitive nerve, its filaments are distributed to the mucous membrane of the larynx, whilst the recurrent branch supplies the laryngeal muscles. In the chest large branches are given off to the cardiac and pulmonary ganglia; and in the abdomen not only does the nerve reach the stomach, but it sends branches to the semilunar ganglia, to the liver, to the pancreas, and to the supra-renal capsules.

The following cases are recorded to illustrate the important influence which pressure upon this nerve exerts in modifying the symptoms of aneurismal disease in the chest.

CASE 1.—*Double aneurism of the arteria innominata; dilatation of the aorta; double bruit; raucedo; dysphagia; paroxysms of dyspnœa; rupture into the trachea; first hæmorrhage five days before death; pressure on the recurrent laryngeal nerve; commencing degeneration of the muscular fibres of the laryngeal muscles.*

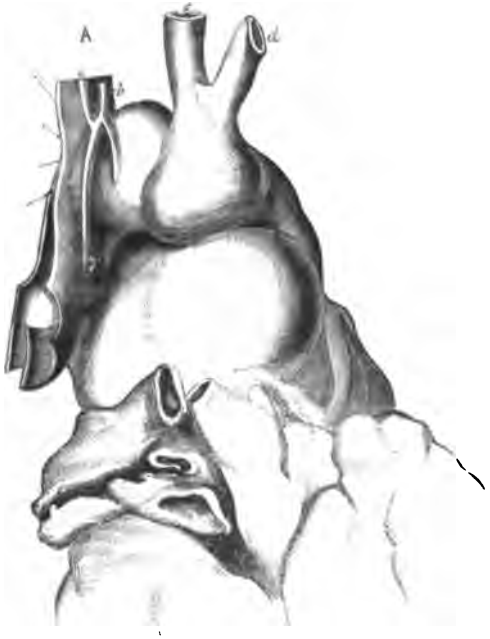
James S—, æt. 39, a strong and muscular man, who had been at work at the Chatham Dockyard, applied to me on November 10th, 1863. In 1854 he went with Sir E. Belcher's expedition in search of Sir John Franklin, and whilst on the Arctic voyage he had an epileptiform attack. He was afterwards occupied in making Armstrong guns at the Woolwich Dockyard, and whilst there, after over-exertion, he had occasionally felt "stupid," and suffered from dyspnœa. The thyroid gland had also been enlarged for a long time.

At the beginning of October, 1863, he suffered from hoarseness, with increased and paroxysmal dyspnœa; the respiration became noisy and stridulous, and he was unable to swallow anything "dry" without producing cough. The sound of the voice varied greatly; sometimes it became comparatively clear and distinct, at other times it was hoarse and scarcely audible; the cough was a slight one. The chest was well developed, but somewhat flattened at the right apex, and the resonance on percussion was imperfect; the respiratory murmur was coarse, but equal on the two sides, and stridulous breathing was heard; both pulses were equal and jerking. The sounds of the heart were normal, but at the upper part of the sternum, towards the right side, a distant, double bruit could be heard. On the right side of the neck the pulsation was greater than in health, but no definite sac could be felt, and no pain was experienced either in the chest or neck, or down the arm. The larynx was carefully examined with the laryngoscope; the epiglottis was seen to be healthy, and the inferior vocal cords were slightly œdematous. Both pupils were rather

smaller than their natural size. On November 30th he came among the out-patients, stating that on the 27th he had broken a blood-vessel, that he had coughed up a considerable quantity of blood, and that the bleeding had not ceased. He was at once admitted into the hospital. Moist crepitation was audible at the right apex, but the bruit previously heard at the right side of the first bone of the sternum could not be detected, being either obscured by the moist sounds or lost from the changed current of blood in the sac. The oozing of blood continued. On December 1st the sputum was rusty, as in asthenic pneumonia, but there was neither faintness nor distress of any kind, and he remained in an easy state till the morning of December 3rd, when, about 2 o'clock, violent hæmorrhage came on, and in a few minutes life had ceased.

On inspection, twelve hours after death, blood oozed from the mouth. The body was that of a strong and muscular man. On opening the chest, the viscera were in their normal position; the lungs were distended with air, and some globules were red, from the presence of blood. Slight pleuritic adhesions existed at the left apex. The larynx was healthy, but the aryteno-epiglottidean folds were œdematous. The trachea and bronchi contained blood, and immediately above the division of the trachea there were four openings, each one capable of admitting a large probe; the openings slightly bulged towards the trachea, and were filled with coagulum, and they extended about one and a half inch in a curved line. The lungs were healthy. The heart was firm and contracted, and its cavities were empty; the mitral and the aortic valves were healthy, but the latter were thin and stretched. The whole of the arch of the aorta, from its commencement, was rough and scabrous, and it was considerably dilated; the arteria innominata was also dilated to three times its natural size, and the right subclavian and the right carotid arose from the upper part of this dilatation; but the current of blood to these arteries did not seem to have

been interfered with. From this aneurismal pouch of the arteria innominata two secondary aneurismal sacs arose; one, about the size of a pigeon's egg, extended downwards and to the right; the other, rather larger, passed downwards and to the left. The latter reached to the side of the trachea and œsophagus, and the rings of the trachea were felt in it. It was this sac which had opened into the trachea; the commencement of the recurrent nerve from the pneumogastric was also upon this inner sac, and the nerve was adherent by tissue. The aneurism had also exerted pressure upon the pneumogastric (see woodcut A). The thyroid gland was enlarged and hy-



a. Recurrent nerve.
b. Pneumogastric nerve.
c. Right carotid artery.
d. Right subclavian artery.

pertrophied. The liver, spleen, kidneys, and intestines, were in a healthy state. On carefully examining the larynx, and especially the inferior vocal cords, the right was more flaccid than the left; on the right side, also, the muscular fibre immediately beneath the elastic tissue was more granular than on the left, and during life it had been more swollen; this state was probably due to the pressure upon the recurrent nerve on that side. It is regretted that the patient was not asked to produce a sound whilst the larynx was being examined.

The occasional cerebral oppression from which this patient suffered might perhaps be correctly referred to enlargement of the thyroid body, and consequent interference with the circulation of the brain, from pressure on the jugular veins; whether this were the case or not, the whole bearing of the general symptoms pointed to aneurism as their cause. The dyspnœa was paroxysmal; the raucedo varied greatly, sometimes there was hoarseness, at other times the voice became clear and distinct; the breathing was to some extent stridulous, and the cough was a slight one. Again, the physical signs tended in the same direction; the absence of bruit immediately over the valves, and the presence of a distant double bruit at the upper part of the sternum, the partial dulness at the same part, without evident mischief about the lungs, were best explained by the aneurismal disease, which had been correctly diagnosed.

The laryngoscope was of value in showing the absence of ulceration of the larynx, and in demonstrating that a further cause must be sought for the dysphagia and the raucedo than could be found in the larynx itself; and although slight œdema of the vocal cords was present, and a difference was detected on the two sides, these conditions were not regarded as the cause of the laryngeal symptoms; and we believe that the value of the laryngoscope in thus facilitating a correct diagnosis will be increasingly found as it is more extensively used. The occurrence of a double bruit in this instance was unusual; frequently no bruit

whatever is heard, and in some instances only a systolic one can be detected. Sometimes, also, a ringing clearness of the normal second sound is the only deviation from the signs of health, as in Case 4. These modifications of auscultatory signs are doubtless due to the different states of the arterial wall, to the amount of fibrin in the sac, and to the course of the blood-current in relation to the sac itself.

There is also great diversity in the severity of the dysphagia arising from aneurism of the aorta; here it was not a distressing symptom; but in some instances, in which the aneurism has taken place on the left side, and when the œsophagus would be likely to be compressed, no such symptom has existed, the tube being pushed aside, *en masse* (see Case 2). On the contrary, the morbid pressure on the œsophagus may lead to sloughing, as in an instance recorded in my work on 'Diseases of the Alimentary Canal,' in which the dysphagia was extreme.

On the occurrence of hæmorrhage in the present instance moist sounds were heard at the apex of the right lung, and this sign led to doubt as to the correctness of the diagnosis. During four days oozing of blood took place from the ulcerative communication between the aneurismal sac and the trachea, and on the morning of the fifth day a sudden increase in the hæmorrhage produced the fatal result. The issue was, however, due in a greater degree to obstruction of the respiration than to mere loss of blood. A much longer period sometimes elapses between the first hæmorrhage and the last, as in a remarkable instance recorded by Dr. Gairdner, and in the fourth case now brought forward blood was coughed up nine months before death.

There was no difference in the force of the respiratory murmur on the two sides of the chest, showing that the sac did not produce direct pressure upon either bronchus; nor was the circulation in the right subclavian interfered with, for the pulses were equal, because the current of blood to the right subclavian artery was uninterrupted. These ine-

qualities of pulse and of the respiratory murmur are, however, valuable diagnostic signs of the presence of thoracic tumours.

The symptoms arising from pressure upon the pneumogastric are modified by the seat of the mischief; in the first instance mentioned the recurrent laryngeal nerve was especially involved, and the symptoms manifested were hoarseness or aphonia, dyspnœa and stridulous breathing, the physical state of the larynx being also altered, as shown by its œdematous condition and by the more granular state of its muscular fibre on one side than on the other; but sometimes this affection of the larynx is much greater, and the laryngeal muscles become pale and wasted; and still further, the pulmonary branches may also be damaged, so as to rouse pneumonic congestion, and even pneumonia. These conditions were remarkably shown in a patient admitted into Guy's Hospital some years ago, but of whose case I only possess a few particulars.

CASE 2.—*Aneurism of the aorta; pressure on the left recurrent laryngeal nerve and on the left pneumogastric; atrophy of the muscles of the larynx, and pneumonia of the left lung; pressure on the thoracic duct; apoplexy.*

George K—, æt. 35, was admitted into Guy's Hospital on March 27th, 1850, and died on April 5th. He was a sailor, and about a month before admission he had an attack of apoplexy; there were also symptoms resembling phthisis, with aphonia; a bruit was heard below the left clavicle, but there was no dysphagia. Dr. Addison diagnosed aneurism. The account of the inspection is principally from Mr. J. Birkett's notes, by whom the examination was made. After mentioning the remains of a large apoplectic clot at the left fissure of Sylvius, around the branches of the middle cerebral artery, he described the state of the thoracic viscera.

A large quantity of sero-sanguineous fluid, with clots of fibrin, existed in the left pleura. The surface of the left

lung was covered with effused lymph of the colour of blood, and from this surface processes passed off, and were attached to the pleura costalis. All these adhesions were recent. The *left* lung was solid; its section presented white softened patches, especially at the apex, and it contained much dirty pus; other portions were studded with numerous white points, resembling cheesy tubercles. The whole appearance of the lung was the result of a low form of pneumonia. The larger air-tubes were filled with thick tenacious mucus. The *right* lung was congested and loaded with serum; otherwise it was normal and free from tubercles; but it was united by old pleuritic adhesions to the walls of the chest.

The heart and its valves were healthy. At the second curve of the arch of the aorta an aneurism had formed, the posterior wall of which was completed by the bodies of the first, second, and third dorsal vertebræ. The bodies of the vertebræ were much absorbed, and the intervening cartilages projected between the bones. The aneurism contained old fibrinous layers, and the walls of the aorta, both above and below, were dilated. The left pneumogastric nerve was stretched out over this aneurismal tumour, and the recurrent laryngeal nerve passed behind the sac in its course upwards. This last nerve was in one part imbedded in the tumour, and its filaments were disorganized; but the nerve between the tumour and the larynx presented the normal appearance. The mucous membrane of the larynx was healthy; but the left crico arytenoideus posticus muscle was pale and small, and indeed atrophied, when compared with the right, which was normal. The trachea was compressed by the aneurism, and the œsophagus was firmly adherent to it; but the latter was pushed aside, explaining the absence of dysphagia.

The liver and the kidneys were congested, and the lacteals in the mesentery were remarkably distinct; they were much enlarged and were filled with white fluid. Although the aneurism was situated near the termination of the thoracic duct, the duct could not be traced.

The pressure on the left pneumogastric nerve had evidently

a causative relation with the pneumonia of the left lung, with the wasting of the left laryngeal muscles, and with the aphonia.

CASE 3.—*Carditis; dilatation of the aorta; fibroid degeneration of the heart; aneurism of the arch of the aorta; pressure on the trunk of the right pneumogastric nerve; pneumonia of the right lung.*

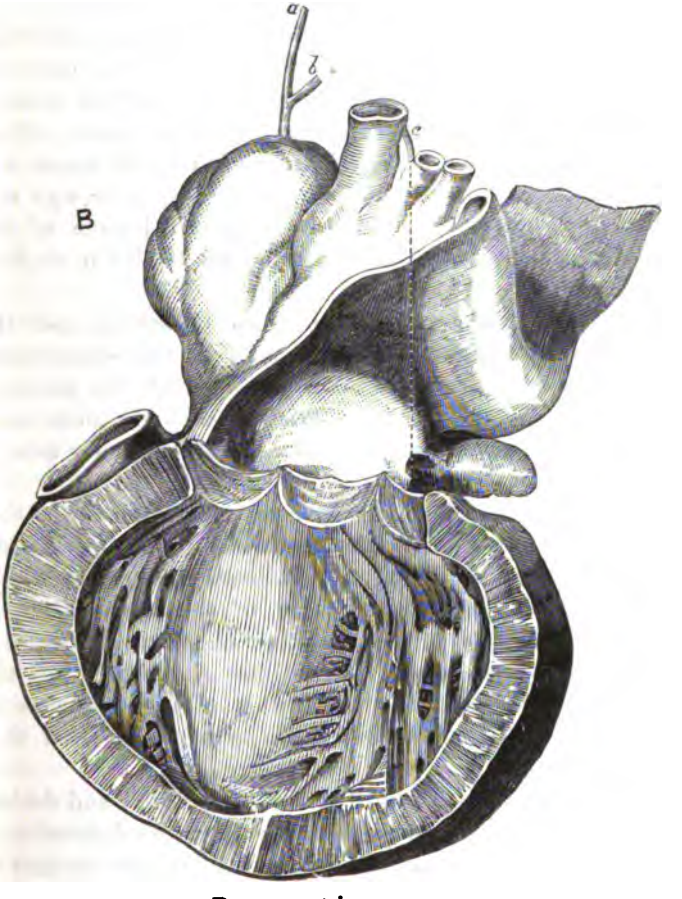
Daniel C—, æt. 45, a groom, was admitted into Guy's Hospital, under my care, October 21st, 1863. He had an aged appearance, was of temperate habits, and had generally enjoyed good health. Two weeks before admission he was exposed to cold and wet, and a few days afterwards cough came on, with urgent dyspnœa. The difficulty of breathing was increased by a recumbent position. On October 21st he had cough, but with very little febrile heat of skin. On examining the lungs, there was no dulness on percussion, but, some râles were audible. The cardiac dulness was considerably increased; the action of the heart was tumultuous, irregular, and intermittent, but a bruit could not be made out; the pulse was small, irregular, and not very compressible. The body generally was spare, the countenance was distressed, and the face slightly congested.

On the 24th the dyspnœa was more urgent, and there was increased dulness about the heart; the action of the heart was still irregular, the sounds were feeble, and at the base of the heart a to-and-fro sound could be heard; there was dulness also at the base of the right lung, and the respiration was there indistinct, and sibilant râles were audible at the right apex. On the left side the respiration was puerile; there was no pungent heat of skin, nor expectoration of blood. There was neither hoarseness nor any difference in the force of the respiratory act on the two sides of the chest. He was cupped from the side to six ounces. Acetate of ammonia, with nitric ether and morphia, were given, and a small quantity of brandy allowed. On the 29th the pulmonary symptoms were greatly relieved, and the action of

the heart became more regular; but a triple sound was now distinctly heard, as if the first sound were divided. The patient continued to improve slowly till November 7th, when symptoms of more general bronchitis came on, apparently from cold taken in the ward; mucous râles were heard on both sides of the chest; there was slight lividity of the countenance, and the dyspnœa was severe; the action of the heart was more feeble. The bronchitis subsided, and the dyspnœa lessened, but several paroxysms of extreme faintness came on at night. On December 1st the legs were found to be swollen, and the urine contained a small quantity of albumen; still he was able to walk about the ward, and, under small doses of tincture of sesquichloride of iron, improved so much that the thought was entertained of his returning home. On December 8th, at 7 p.m., faintness again came on, and he passed a disturbed night. On the following morning the faintness continued, and he rapidly sank.

Inspection, on the 10th December.—There were pleuritic adhesions on both sides. The whole lower lobe of the right lung was red, fleshy, and dense, and some lobes were still more congested. *Heart.*—There were partial adhesions over the pericardium, and upon the ventricles, and small portions of fibrin were attached to other parts of the serous membrane. The heart was much enlarged; the auricles and right ventricle were normal, the left ventricle was hypertrophied and dilated; the endocardium was white and opaque in several parts, especially over the carneæ columnæ of the mitral, and on making a section of these muscular columns a considerable quantity of fibroid deposit was seen. This fibroid deposit was still more manifest in microscopical examination, fibre-tissue being observed between the bands of fibre; but the muscular fibre itself had not undergone degeneration. The membranous portions of the aortic and mitral valves were healthy. The aorta was scabrous and dilated, and at the commencement of the transverse arch, extending posteriorly, and towards the right side, was an aneurismal sac about two inches in diameter; it extended to the apex of the right lung, but did not perforate the tissue. The sac

was filled with fibrin. The right pneumogastric nerve extended *upon* the sac, but the recurrent nerve was given off *before* the trunk reached the aneurismal tumour, and this fact—the pressure of the trunk of the right pneumogastric—appeared to explain the presence of the pneumonia on the right side, whilst there was no affection of the larynx. The liver and kidneys were healthy, so also the other viscera.



a. Pneumogastric nerve.
b. Recurrent nerve.
c. Branch of sympathetic nerve.

A large branch of the sympathetic nerve was found upon the posterior aspect of the distended aorta.

In this case we had no history of rheumatism to which the carditis would be attributed, and we were compelled to refer the disease to excessive muscular exertion. There was evidence that both the pericardium and the endocardium had been affected, and the mischief had extended into the muscular fibre. On admission into Guy's, the disease had become aggravated by bronchitis, and it seemed probable that there was some pericardial effusion. Dilatation, with the degenerated condition of the muscular fibre, led, we believe, to the irregular and tumultuous action of the heart. Afterwards, the irregularity diminished, and a triple sound was heard, as if the first sound were divided. This sign was referred to degeneration of the carneæ columnæ of the mitral, and the post-mortem examination tended to confirm the idea.

Exposure to the weather produced bronchitis, and the congestion of the lungs was increased by the embarrassed condition of the heart; but we believe that the pressure on the trunk of the pneumogastric nerve by the aneurismal sac led to the pneumonic consolidation of the lower lobe of the right lung.

Another interesting symptom in connection with this case was the extreme faintness which came on during several occasions at night; these attacks were probably due to the dilatation and degeneration of the heart with pericarditis; but one of the large cardiac nerves was also involved in dense tissue on the posterior aspect of the distended aorta, and the implication of the branches of the sympathetic nerve may have greatly increased this symptom.

The aneurismal sac was distended with fibrin, and during life did not produce *any physical sign*, but the distension of the nerve-structures and the impediment to the current of the blood increased the syncope and the embarrassment of the heart.

CASE 4.—*Aneurism of the aorta; feeble voice; pressure on the recurrent nerve; dysphagia; intense pain; perforation into the trachea.*

Gerhardt B—, æt. 57, was admitted into Guy's Hospital, July 1st, 1863, under the care of Dr. Gull. He was a German, but had lived for about sixteen years in England, as a shoemaker. When thirteen years of age he had had rheumatism; but his present illness commenced about six months before admission, with symptoms of simple catarrh, which increased to a cough about the middle of March, 1863. Blood was expectorated, but the hæmorrhage ceased for several weeks. He was of dark complexion; the countenance was distressed, and the respiration was difficult, as if there were obstruction of the trachea; there was also general pain in the chest, from the scrobiculus cordis to upper part of the sternum, and round to the back. The tongue was furred, red at the edges; pulse 80, and feeble; the bowels confined. There was dulness at the right apex posteriorly, with coarse bronchial breathing; the other parts of the chest posteriorly were resonant. On the right side in front the respiration was coarse and loud, and there was great fulness of the veins; dulness existed at the right sterno-clavicular articulation, but no abnormal pulsation could be felt; the radial pulse was feeble on both sides; deglutition was difficult, and he could neither lie upon the back nor upon the left side. He was ordered brandy in small quantity, with chloric ether ℥x, tincture of opium ℥v, in camphor mixture, every six hours, and castor oil if necessary. On the 6th he had pain, especially in the right shoulder; deglutition was difficult, and it was accompanied with pain in the larynx. There was a soft swelling on the right side of the neck, beginning just above the clavicle, and disappearing under the sterno-mastoid muscle; pulsation was stronger on the right side of the neck than on the left. On the 10th the motion of the left upper costal cartilages was less perfect than on the right side; the second sound of the heart was increased; the

voice was deep, but distinct; the pulsation of the right carotid could hardly be felt in the lower part of the neck, but it was distinct on the left side. He felt pain round the lower part of the chest to the right axilla. When left to himself, the patient rested on his face, for in that position the expectoration of blood was lessened. The blood was of a dark colour, resembling venous blood. Ordered dilute sulphuric acid, with tincture of opium. On the 20th he was able to sit up; the supra-clavicular spaces were depressed at each inspiration, and so were the intercostal spaces, especially at the lower part of the left side; there was also fulness at the lower part of the right sterno-mastoid muscle. The most severe pain was now experienced in the left axilla, less on the right, and still less at the epigastrium. On the 21st he felt severe pain in the left shoulder, and down the left arm to the back and front of his hand and fingers. On the 23rd the pain was still more severe. For a few days he became more comfortable, but at the beginning of August the dyspnœa increased, so also the dysphagia and the pain. The voice was very weak, but deep and distinct, as if he were whispering. The second sound of the heart was also preternaturally distinct over the first bone of the sternum, and accompanied with a strong impulse; the right radial pulse was rather stronger than the left; from noon till midnight his distress increased; after midnight it lessened. The hæmorrhage had now ceased. The symptoms varied in severity till October 26th; thus, at one time he could rest upon his back, at another paroxysms of urgent dyspnœa came on, and he was compelled to rest upon his face and knees to obtain any relief, supporting his head with his hands; the hands then became hard and cold, and the body trembled. On December 5th double pulsation could be felt immediately above the sternum, the dyspnœa was urgent, and the patient called the expectorated matters "rotten blood." For several nights the respiration had become extremely difficult, and on the 13th he fell from his bed and fainted. At 2 a.m. of December 14th he had a slight attack of hæmorrhage, the blood being mixed with

tenacious mucus, and he sank on the evening of the same day.

The inspection was made by Dr. Wilks. The larynx was apparently healthy. There were pleuritic adhesions at the apex of the left lung. The lungs were partially distended with air, and the trachea and bronchi contained blood. There was no pneumonia. The trachea was greatly compressed immediately above the bifurcation, and it was contracted almost to a fissure; the mucous membrane was irregular, and a small opening was seen, through which a probe could be passed unto the aneurismal tumour, and through which blood had escaped. The heart was healthy, and the valves were normal. The aorta was rough and scabrous. At the termination of the arch of the aorta was an aneurismal sac, the size of the fist; it extended upwards, backwards, and to the left; it reached the trachea and œsophagus, and had compressed them, especially the former, and blood had escaped through the opening previously mentioned. The sac was lined with firm fibrinous layers, so that the rapid discharge of blood had been prevented. The recurrent laryngeal nerve was found greatly stretched and closely imbedded in the posterior wall of the sac. Other viscera were healthy.

On comparing this case with the preceding, the great diversity which exists in the symptoms of thoracic aneurism is apparent in a marked degree. In the former the indications of disease arose especially from the state of the heart; no pain was complained of, no abnormal pulsation could be felt, and no dysphagia existed; in the latter the pain was agonising, the dyspnœa most urgent, and the dysphagia distressing. The pain was especially in the course of the intercostal and of the brachial nerves, and arose from direct pressure. The contraction of the calibre of the trachea led to the dyspnœa; but it was increased and rendered of a more intense and paroxysmal character by the pressure upon the recurrent nerve; the voice was weak and whispering from the same cause. The pain and distress were even

greater than in some instances in which the bodies of the vertebræ have been absorbed, and the case tends to confirm the statement of Dr. Stokes—that pain does not necessarily arise from affection of the vertebræ. The long period of time during which the oozing of blood continued might easily have led to the belief that the tumour was non-aneurismal. The hæmorrhage commenced nine months before death, and ceased sometimes for several days; this slow discharge was explained by the manner in which dense layers of fibrin had covered over the opening into the trachea. The heart was healthy; but Dr. Gull was led to suspect aneurismal disease rather than simple tumour from the ringing character of the second sound, associated with the increased pulsation above the sternum.

The recurrent nerve on the left side was closely connected with the walls of the aneurism, and a more marked affection of the larynx might have been anticipated than was expressed by the feebleness of the voice and dyspnœa; no degeneration was found in the minute muscles of the larynx itself. This leads us to notice another effect of pressure or irritation of the recurrent nerve—*spasmodic contraction* of the larynx, and the paroxysms of dyspnœa were probably greatly aggravated in the last case by this cause. An instance of *fatal* dyspnœa, apparently spasmodic, from aneurismal disease of the aorta, is recorded in my work on 'Diseases of the Abdomen.' The patient had slight dysphagia, with paroxysms of urgent dyspnœa; the respiration in the left lung was rather less distinct than in the right, and the voice was scarcely changed. He died in one of these paroxysms, a few moments previously having been usefully employed in the ward; and no cause for the fatal apnœa could be found, except the pressure of the aneurismal sac upon the recurrent laryngeal nerve. Thus, pressure on the pneumogastric nerve and its recurrent branch in the chest produces symptoms according to the seat of pressure. (1) If the recurrent nerve be irritated, spasmodic contraction of the laryngeal muscles takes place, and with modified voice there is more or less paroxysmal dyspnœa, as in Case 4;

but (2) if the recurrent nerve be more completely compressed, it leads to degeneration, wasting, and loss of muscular power, in the same muscles, and aphonia, paroxysmal dyspnœa, and cough, are induced (Cases 1 and 3). Again (3) if the recurrent nerve be uninjured, the aneurism being below that nerve, and the trunk of the pneumogastric is compressed, then, whilst the voice and larynx are unaffected, congestion of the lungs and broncho-pneumonia are more likely to arise. Still, those parts only are changed of which the nerve supply is directly modified. When the recurrent nerve is injured, symptoms of disease are manifested *only* in the larynx; and when the trunk of the nerve is compressed at a lower site, whilst the larynx escapes and the voice is unaffected, the nutrition of the lung is changed.

We do not find, in pressure on these nerves from aneurism of the aorta, the radiation of irritation as in disease implicating the peripheral branches. Thus, the superior laryngeal nerve, having its origin in the neck, does not suffer, and its branches to the mucous membrane of the larynx and epiglottis show no symptom of disease; the pharyngeal branches of the same nerve are also unaffected; and in those instances where the auricular branch of the pneumogastric has apparently been involved, producing pain in the ear, it has probably been a coincident circumstance.

In *peripheral* irritation of the branches of the pneumogastric nerves, whether that irritation be in the lungs or in the stomach or in the supra-renal capsules, a more general sympathy may be traced. The gastric branches of the nerve are generally affected, and vomiting, with other abdominal symptoms, are caused by tubercles in the lungs. But although these gastric symptoms are frequently observed from irritation of the peripheral branches in the lungs, especially in early phthisis, and equally so in disease at the cerebral centre of the nerve, still I have never witnessed marked gastric symptoms from aneurismal disease of the thoracic aorta. The pain which is complained of, as in the fourth case, at the scrobiculus cordis, was due to direct irritation of the intercostal nerves, and it did not arise in that

instance from the stomach. Still, since the stomach is situated below the seat of pressure, it may very naturally present modified action, as in an instance recently recorded by Dr. Hughlings Jackson.

The difference in the symptoms of thoracic aneurism, according to the implication or freedom of the pneumogastric and recurrent nerve, may be used in diagnosing the precise seat of the aneurism, remembering, however, that direct pressure upon the trachea and bronchi may also modify the symptoms.

The pneumogastric nerve in the chest supplies also the œsophagus, and this canal is affected in an almost similar manner as the larynx and trachea, by alteration of its nerve supply. For not only do we find that it is directly pressed upon, and severe dysphagia produced by diminution in the size of the canal, so that sloughing takes place, but spasmodic contraction of the muscular fibres and changed nutrition result from pressure on the œsophageal nerves. The dysphagia in aneurism, like the dyspnœa, is paroxysmal, and in one instance of thoracic aneurism we found ulcerative communication between the œsophagus and bronchus, produced, apparently, without direct pressure. The patient had an aneurism at the commencement of the aorta, and perforation took place into the pericardium; a second smaller aneurism existed below the subclavian; but it did not appear to exert any direct pressure upon the œsophagus, so as to cause the ulcerative opening into the bronchus.

Cancerous disease of the œsophagus, implicating the pneumogastric nerve, leads to similar spasmodic contraction of the canal in a remarkable manner, and in many instances, as we have elsewhere shown, to disease of the lung itself, to broncho-pneumonia, or even to sloughing.

As to the effect of the pneumogastric-nerve pressure upon the heart, it must be borne in mind that where the action of the heart has been greatly disturbed, disease, either of the valves or of the muscular fibre, has been generally found; and in some cases it would seem that the large branches of the vaso-motor nerve have had to do with the

enfeebled action of the heart and with syncope, as well as with sensations of cerebral pulsation, with contraction of the pupil, noises in the ears, and other allied conditions.

The cause of the aneurismal disease in these four instances, all men, was probably violent muscular exertion. There had been the constant recurrence of strain upon the aorta, and the first part of the vessel had become dilated. The lining membrane of the aorta was, in all, rough and scabrous; but the aortic valves had so far resisted the strain upon them, that no regurgitation had taken place, and they were, in each patient, in a healthy state; neither was the left ventricle greatly hypertrophied, as we find in aortic regurgitation. In many instances it is true that the aortic valves at length become diseased, from the long-continued strain; but the remark of Dr. Stokes is generally borne out—that valvular disease is rare in aneurism of the thoracic vessels; and in the third case recorded, in which the symptoms of cardiac disease were more evident than the aneurismal, it was the muscular fibre of the heart, and its investing and lining membrane, which were diseased, rather than the valves.

Other nerves beside the pneumogastric become affected by direct pressure in thoracic aneurism. Sometimes the phrenic is compressed, but more frequently the first dorsal nerve is implicated, and there is pain in the course of its branches. This latter nerve receives a large branch from the sympathetic ganglion, and thus becomes intimately connected with the cardiac branches upon the aorta. In the fourth case the phrenic nerve passed in front of the sac, but it did not appear to have been compressed, and no symptom could be traced to its source.

ON THE

ENDEMIC HÆMATURIA

OF THE

CAPE OF GOOD HOPE.

BY

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COMMUNICATED BY

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Endemic hæmaturia is a disease indigenous to hot countries. In these temperate regions we are, happily for us, altogether free from it. Medically speaking, there is but one drawback to this immunity—we are almost entirely precluded from searching into its cause and pathology. When, therefore, a case of endemic hæmaturia comes under our observation, it cannot fail to excite our interest and engage our careful attention.

Three months ago, a robust gentleman, between twenty and thirty years of age, a resident of the Cape of Good Hope, consulted me about a slight ailment. On his second visit he mentioned incidentally that for several years past he had frequently passed a little blood with the urine. He said this disease was very common in the Cape, in some parts of

which both men and women suffered from it. He mentioned Uitenhage as being a place in which it was particularly prevalent. It was commonly supposed there to be a gravel complaint, caused by drinking the water, which, he informed me, was derived from a good clear spring, arising nine or ten miles distant, in Winterhoek Mountain, and conveyed, partly through bush, in an open channel cut in the red ferruginous clay. The water thus brought to the town is distributed through the streets in open gutters, which are paved in some parts, but in others the bed is alluvial, and allows of the growth of numerous water plants. The drinking-water of Uitenhage is obtained from these gutters; horses and other animals have ready access to them, and by means of diverticula, the gardens are watered from them.

Having described to me the symptoms of his disorder, which I have detailed below, I requested him to send me some of his urine for examination, and while I was awaiting its arrival I made some further inquiries from my Cape friends and acquaintance concerning the hæmaturia of this part. Messrs. Edwin and G. D. Atherstone and Mr. Walter Mills corroborated the statements of my patient. Those of their schoolfellows, they told me, who came from Uitenhage all voided blood in the urine; it was a common complaint there, and was supposed to be caused by drinking the sandy water which flows through the streets.

From the foregoing evidence it is clear that there exists in some parts of the Cape an endemic hæmaturia, the cause of which is at present unknown.

Thirty degrees east of Uitenhage, and ten degrees nearer the equator, in Mauritius, hæmaturia is known to be endemic. Chapotin,[1] Salesse,[2] and Rayer,[3] have each recorded several cases of the disease existent in the Isle of France. Dr. Todd, in his work on 'Urinary Diseases,'

[1] Chapotin, Ch., 'Topographie médicale de l'Ile-de-France,' in-4, Paris, 1812.

[2] Salesse, Antoine-Emilien, 'Diss. sur l'hæmaturie ou pissement de sang,' in-4, Paris, 1834.

[3] Rayer, 'Maladies des Reins,' vol. iii.

page 62, makes the latest mention of it; he says, "Hæmaturia is so common in the Isle of France that few, I am informed, of the male population escape it." One young man, who had resided there for five years, came under his treatment for the disease. "The blood was always small in quantity, never so much as to discolour the great bulk of the urine; it came with the last portion only, quite at the end of micturition, a few drops of apparently pure blood escaping at the last. Sometimes small clots were discharged, without any definite shape. After standing, the urine would deposit a sediment of a whitish or reddish mucus, composed of bladder-epithelium, crystals of oxalate of lime, and a few small cells, which presented all the microscopical characters and actions of those of pus- and blood-corpuscles. There was no appearance of casts of tubes or of renal epithelium. The source of the hæmorrhage," he continues to say, "was doubtless the bladder, and the disease seemed to be essentially a catarrh of that organ, with occasional hæmorrhage."

I will now enter into the details of the case which has come under my own observation.

Previous history.—About four and a half years ago my patient had the "low fever" of the Cape, and afterwards took a journey to Japan to recruit his health. He remained in this island three weeks, and dwelt during the whole of the time at Nagasaki. The drinking-water, he remarked, was dirty and disagreeable. A fortnight after leaving Japan, and while on board ship, he first noticed symptoms of hæmaturia. On his homeward journey he visited China, and remained there six weeks.

Symptoms.—The first indication of the disease was the passage of a little blood after emptying the bladder of clear urine; the blood was dark coloured, and amounted usually to a few drops; it never exceeded a teaspoonful. For the first fortnight it appeared after every act of micturition; it has continued, with weekly or fortnightly intermissions, ever since—a period of about four years. Unusual exercise and railway travelling always cause a slight increase in the quantity of the blood. For the last year the bleeding has

been frequently substituted by the passage of soft but consistent filaments, irregularly cylindrical, sometimes nearly colourless, but usually deeply blood-stained. They occasionally interrupt the flow of urine for ten or twelve minutes, but are then voided, and the obstruction is removed. Excepting the last table-spoonful, the urine itself is never coloured by blood. During the last few months, and when in a state of quietude, these red threads, or "veins," as he calls them, have altogether taken the place of blood. There is no increased frequency of micturition, no irritability of the bladder, but the desire to micturate is a little stronger than usual, and generally requires immediate attention. The quantity of urine voided is natural. There is no tenderness in the pubic or lumbar regions, but a sudden and sharp twinge of pain is occasionally experienced in the loins, such as, if it come on during a walk, necessitates a halt for a few minutes. It subsides as suddenly. The complaint causes but little annoyance, and the health is otherwise very good. He was travelling in company with a gentleman resident in Uitenhage, who was then suffering from precisely similar symptoms.

Condition of the urine.—In the early part of October, 1863, I received six ounces, which, on rising in the morning, had been passed directly into a clean bottle. Pale-amber coloured, specific gravity 1017·6, acid, deposits a deep layer of dirtyish-white flocculent matter, amongst which were two short opaque filaments about the $\frac{1}{35}$ of an inch in diameter, of a brownish colour and soft consistence, two shorter and wider fragments of the same substance, a little reddish mass the size of a hemp-seed, like a little clot of blood, and numerous white specks. The clear limpid urine, when acidulated with HO,NO_5 and heated, deposited a trace of albumen.

The secretion was examined from time to time, and presented little or no variation. It was usually pale and whey-like, deposited the flocculent matter, furnished a trace of albumen, and contained the filamentous bodies and specks. Sometimes the specimen was loaded with

fine crystals of uric acid and urates; oxalate of lime was never altogether absent—in most samples it was abundant; sometimes there was a little blood-stained, slimy mucus.

Abnormal constituents of the urine.—Except the albumen and crystalline deposits above mentioned, these were composed of pus-corpuscles, which, with the fine amorphous deposit of oxalates, formed the flocculent matter, a few blood-corpuscles; and the filamentous bodies and coagula. It is these latter which will now engage our attention. Examined under a half-inch object-glass, they were found to be composed of round or flattened, often branched masses of mucous cells and soft molecular mucous fibres, forming together a firm coherent matrix, more or less stained with blood, and imbedding a variable number—sometimes three or four, sometimes thirty or forty or more—bright, highly refractive, oval bodies (Plate II, fig. 2), which I had no difficulty in recognising as the ova of some entozoon. The mucous casts were sometimes dirty white, and composed wholly of mucus; sometimes they contained a considerable number of red blood-corpuscles. They varied much in diameter, the smallest measuring about the $\frac{1}{500}$ of an inch. The larger were generally flattened, the smaller were cylindrical. *a*, Fig. 1, are all the filaments and coagula which were contained in one sample of urine; *b*, Fig. 1, all those from another.

Ova.—Composed of the immature embryo contained in the egg-case, elongo-ovate, $\frac{1}{170}$ inch in length, $\frac{1}{400}$ inch wide, being about the same size as the advanced eggs of the cheese mite (*Acarus domesticus*); anterior extremity acuminate. Spine $\frac{1}{6000}$ inch long, sometimes straight (Fig. 6), sometimes deflexed (Figs. 7 and 12), the base usually confused with the rest of the egg-case (Fig. 6), sometimes abrupt. Egg-case a bright hyaline, chitinous envelope, unaffected by alkalies and acids, the $\frac{1}{10000}$ of an inch thick, presenting a double contour line, and dehiscing longitudinally (Figs. 4, 8). Contents enclosed in a distinct vitelline membrane, and composed of a solid mass of clear spherules and granules (Figs. 3, 7, &c.). The former average the $\frac{1}{4500}$ of an inch in diameter. The largest are usually aggregated about the

centre and anterior extremity of the embryo mass. After maceration in water slightly acidulated with HCl, the albuminous constituents appear to be dissolved and the interior resolved into a mass of strongly refractive spherules of fatty matter (Figs. 8 and 9). Generally no organs can be distinguished. Fig. 6 represents the nearest approach to organization that I have observed. In this ovum the anterior extremity of the embryo mass forms a papillary projection, and there is an appearance of lines and of two little pyriform bodies converging to it. Here and there a minute cell or two is observed to lie between the egg-case and the investing membrane of the embryo. Sometimes the ova are much elongated, and when this is the case they either remain straight or become elegantly curved, their opposite extremities being waved in different directions (Fig. 12).

In one branched mass of mucus the ova appeared to be in a more advanced stage of development, the ovisacs dehiscing very readily on pressure, and liberating the immature embryo, as is represented in Fig. 4. It is broader and less symmetrical than the egg-case, is attenuated at one end, but as yet there is no apparent differentiation into distinct organs. Numerous dehisced and empty egg-cases lay in the mucus.

Mature embryo.—Outside another mucous cast, and entangled in the meshes formed by its branches, I was fortunate enough to discover several free mature embryos (Figs. 10, 16). They measure $\frac{1}{300}$ to $\frac{1}{160}$ of an inch long, and the $\frac{1}{350}$ inch broad, being a little larger than the egg-case. The general shape is elliptical, but the sides are rarely symmetrically curved; the posterior extremity is a little contracted and rounded; the anterior terminates in a papillary sucker-like prolongation, possessing a central depression leading to a canal into which two or three smaller canals appear to converge. These canals are lost below in the spherules which are contained within the interior, or in a pyriform mass of them, which is sometimes observed to occupy the anterior part of the embryo. Besides this there is in some of the embryos indications of a

differentiation of the interior, which is composed of granules and spherules of various sizes, partially distributed. The whole external surface, which is formed of a distinct thickish integument, is clothed with delicate, exceedingly close-set cilia; they are best developed at a little distance from the sucker-like anterior extremity, where the surface is minutely punctated by the origin of the cilia. Fig. 15 shows the escape of a mature embryo from the egg-case.

Seeking to get some knowledge of the animal in the more advanced stages of its existence, I have met with a small portion of ciliated integument, which, as it may belong to the parasite, I will briefly describe. The minute fragment is delineated in Fig. 11. From its rounded form I think the individual to which it belonged was more spherical than cylindrical. The cilia or hairs are simple, homogeneous, and elongato-conical; they average the $\frac{1}{300}$ of an inch long, and are distributed at intervals of the $\frac{1}{800}$ of an inch. The integument itself is delicate and homogeneous, or only very faintly granular. The relative sizes of this bit of integument and the mature embryo may be judged of by comparing Figs. 10 and 11, bearing in mind that Fig. 10 is magnified thrice as much as Fig. 11.

So far for facts. I have now to inquire to what particular animal the parts above described belong. They have no relationship whatever to the cestoid entozoa; the form and structure of the egg and hooked embryo in this class are quite different. The anatomical characters of the ovum, its development into a ciliated embryo, the form of that embryo (which is that of an adult Distomum) and the probability, suggested by the piece of ciliated integument—which, in the absence of other means of diagnosis, I may fairly, I think, use—that the adult parasite possesses a ciliated integument, all point to the trematode worms. The ciliated embryo closely resembles a certain species of Monostomum, and it possesses also several characters in common with the embryos of such of the Distomata as have been described. Monostomum does not, as far as is known, inhabit the human body; I shall therefore limit

myself to a comparison with the genus Distomum. Six species of this genus inhabit man, viz., *D. hepaticum*, *crassum*, *lanceolatum*, *heterophyes*, *hæmatobium*, and *ophthalmobium*. Too little is known of the last-mentioned species to allow of any comparison. The ovum and ciliated embryo above described are quite distinct from the corresponding parts both of *D. hepaticum* and *D. lanceolatum*, and probably also from those, which have not yet been observed, of *D. crassum*. The eggs of the former two species are wanting in the anterior spine; the ciliated embryo of *D. hepaticum* is conical, and the papilla which terminates its anterior extremity is depressed. The ciliated embryo of *D. lanceolatum* is oblately spherical, and ciliated only about its anterior extremity.[1] We may therefore confine our attention to the two remaining species. *D. heterophyes* is very minute; its length does not exceed three fourths of a line. The skin is beset with small spines, directed backwards, and they are particularly numerous in front. The intestinal canal is composed of a short, narrow, membranous œsophagus, terminating in an oblong muscular pharynx, which is continuous with a narrow cibarian canal, dividing, as usual, in front of the ventral sucker into two lateral intestinal tubes.[2] This rare parasite was discovered by Dr. Bilharz in Egypt. He found them in two cases only, inhabiting the small intestine in great numbers. But it is to *D. hæmatobium* (*Bilharzia hæmatobia, Gynæcophorus hæmatobius*) that the organisms in question have the greatest resemblance. Bilharz,[3] and Griesinger,[4] followed by Derseble,[5] Kückenmeister,[6] and Leuchart[7] have described this parasite. It is a white, soft-skinned, elongated entozoon, resembling a nematoid worm. The anterior part of the body is smooth, the posterior part

[1] See figs. in Leuchart's 'Die Menschlichen Parasiten.'
[2] Bilharz and Siebold, 'Zeitschrift für Wissenschaft Zool.,' iv.
[3] Ibid., § 59, 72, and 454.
[4] 'Archives für physiol. Heilkunde,' 1854, Jahrg. xiii, § 561.
[5] 'Wiener medic. Wochenschrift,' 1856, N. 4.
[6] 'Manual of Parasites,' vol. i.
[7] Op. cit., p. 491.

is beset with short hairs; its length is three lines; it has an oval and a ventral sucker, and a gynæcophoric canal. The ova are strongly pointed at one end, the egg-shell dehisces longitudinally. The ciliated embryo measures 0·37 millimètre, $= \frac{1}{67}$ of an English inch, in length, and the 0·11 millimètre, $= \frac{1}{227}$ of an inch, in breadth. It has a cylindrical form, and is rather thicker anteriorly than posteriorly. It is covered all over with tolerably long cilia, and is furnished at the anterior end with a proboscidiform projection, presenting a sucker-like impression from which run two slender lines, terminating in a pair of closely approximated sacs. The presence of the gynæcophoric canal is a sufficient reason for separating this animal from the genus Distomum; I shall therefore follow Dr. Cobbold, and call it, after its discoverer, *Bilharzia hæmatobia*.

The resemblance of the parts above described with the corresponding parts of *Bilharzia hæmatobia* will now be readily inferred, and they will be found to be very close indeed. The two parasites further agree in other important particulars.

B. hæmatobia is the cause of the endemic hæmaturia and gravel complaint, or lithiasis, of Egypt, and the parasite is so abundant in this country that Griesinger found it in 117 out of 363 human post-mortem examinations. It chiefly inhabits the small veins of the mucous membrane and substance of the urinary apparatus.

After careful comparison, however, of the ciliated embryo, which I have described, with Griesinger's figures of *B. hæmatobia*, there remain sufficient differences to induce me for the present to refer the former to some other species; and from the locality in which I have discovered the parasite, I will call it *Bilharzia Capensis*.

With respect to the distribution of *Bilharzia*, it is worthy of remark that Cairo and Uitenhage of the same continent are equidistant, north and south, from the equator, and within 15° of the same longitude. I ought, however, to mention that my patient, who is a very observant man, believes he derived the urinary disease from the water in use at Nagasaki; but, according to this supposition, the animal

must have arrived at mature development, and have manifested its presence in the urinary organs, within four months. I am rather inclined to believe that he received the animal at the Cape.

It were unsafe to conclude from this single instance of the association of the parasite above described with hæmaturia, that it is the constant cause of the hæmaturia of the Cape, but from what we know of endemic hæmaturia the probabilities are greatly in favour of this conclusion.

Since writing the above I have received, through my friend, Mr. E. Atherstone, an introduction to Mr. George Dunsterville, F.R.C.S., surgeon to the infirmary at Port Elizabeth. This is a singular piece of good fortune, for Mr. Dunsterville has been in practice in that town for twenty-seven years, and is therefore familiar with the disease.

Mr. Dunsterville has most courteously furnished me with the following particulars:

1. Hæmaturia is common in Uitenhage and in Port Elizabeth. It has been long prevalent in the former town. The female sex and the native population, as far as Mr. Dunsterville is aware, are free from it. It affects boys at the age of three or four years, and is most prevalent between this age and sixteen. Two, out of every three schoolboys, are affected with it. It gradually disappears about the age of puberty.

2. The symptoms are a smarting or burning sensation in passing urine, and at the end of micturition a flow of about a teaspoonful of pure blood, or a discharge of ropy mucus tinged with blood. The shirts of boys affected with the disease are often stained as if they had the menstrual discharge of the other sex. There is no pain in the urinary organs. The urine is never stained with blood. Gravel is a common complaint, stone is not uncommon.

3. Of the cause of the disease nothing was known. It was so common, occasioned so little inconvenience, and fairly exhausted without avail every plan of treatment; while, at the same time, experience showed that it generally subsided after the age of puberty, and apparently entailed

no subsequent complications, that the medical practitioners, in the absence of proper means and sufficient leisure for making minute investigations, had long ceased to pay particular attention to the disease.

4. The drinking-water in Port Elizabeth is partly derived from the superficial drainage of twenty miles of plain by means of wells ten or twelve feet deep, and partly from rain water conveyed by zinc or iron roof-gutters into tanks of various material. The well water is brackish.

Almost the whole of the vegetables, including salads, used in Port Elizabeth are obtained from Uitenhage. Periwinkles are very abundant in Algoa Bay.

The natives and colonists are affected with Tænia and *Ascaris lumbricoides*, both of which parasites are very common. Some time ago the latter appeared in great numbers, and infested both old and young. Some patients evacuated during this epidemic as many as forty worms.

A year ago a number of horses died from an epidemic of "bot worms," which attacked the stomach.

Mr. Dunsterville kindly introduced me to his two sons, young gentlemen of the ages of seventeen and twenty respectively. They had, in common with the other young men of Port Elizabeth, suffered from hæmaturia, but it had disappeared of late, and they considered themselves to be free from the disorder. The elder (Mr. G. Dunsterville, jun.), however, had been greatly troubled during the last six months with gravel, and had passed during this time four or five renal calculi. He described his urine as being like chalk and water. On examining it, I was able to demonstrate to Mr. Dunsterville the presence of several ova of Bilharzia. One is delineated in Fig. 13. The secretion was highly acid, and loaded with a fine crystalline deposit of oxalate of lime and uric acid.

The urine of the younger gentleman (Mr. C. Dunsterville) was full coloured and clear, but usually contained a little deposit of oxalate of lime in distinct octahedra. In the first sample which I examined I could detect with a pocket lens a few floating ova, which under the microscope presented all

the characters of those above described. Besides these I found a long sinuous tube (Fig. 14), dilated at one end into a pyriform enlargement (*a*), about $\frac{1}{50}$ of an inch long and $\frac{1}{150}$ of an inch wide; another similar dilatation of half the size (*b*) occurred about the middle of the length of the tube. At the extremity of the first dilatation or sac, and apparently attached to it, was a third pyriform sac (*c*), corresponding in size to the second. From this, two secondary tubes, smaller than the primary one, parted, the one (*f*) being continuous with it below; the other (*g*), which was indistinctly connected with it, passed away for a distance, and, after apparently joining the first, terminated in a free extremity. The main tube was considerably contracted at a distance from the second dilatation, and around this part, and apparently connected with it, were a number of excessively fine linear processes (probably tubes), some of which were very long (*d*); below this the tube dilated into a wider terminal portion (*e*). The length of the extended tube was about one sixth of an inch. The whole organ was structureless and of the most excessive delicacy; it contained only a few bright granules. It is probably the intestinal canal of the parasite.

I have repeatedly examined (during December) the urinary secretion of both these gentlemen, and have never failed to detect ova; generally they were numerous, and some hundreds must have been passed most days. When the crystalline deposits were abundant, the ova were usually encrusted with them.

Mr. G. Dunsterville, jun., has suffered much of late from the passage of small renal calculi, and has brought me several for examination. They are of a dirty white colour and crystalline structure, and are chiefly composed of oxalate of lime. On crushing them and treating them successively with nitric acid and solution of potash, I was enabled to detect the presence of considerable numbers of ova, which had, no doubt, afforded a nucleus for the crystalline deposit. In this case the symptoms indicate that the parasite inhabits the kidney.

It is remarkable that the hæmaturia should thus disappear while its original cause still remains. It can only be accounted for by supposing that the animals form non-vascular cysts around them, which retain communication with the natural passages.

The parasitic origin of the hæmaturia of the Cape being thus satisfactorily, I believe, determined, I would say a few words on—

Treatment.—Mr. Dunsterville tells me that every remedy which exists for hæmaturia has been tried to the full without effect. But it must be borne in mind that the medicines were administered empirically, and with a view to check a symptom, and not to remove the cause, which, being now known, we may at least hope to prevent.

In the treatment of the disease our efforts must be directed—(1) to kill or expel the adult sexual parasites; and, should our efforts to do so be unavailing, (2) to secure the regular expulsion of the ova, which, so long as they remain in the body, may at any time become the nuclei of urinary calculi. It has been already stated that gravel is very prevalent in the same localities as the hæmaturia; and, as I have shown in one case, there can be very little doubt that it is caused in the majority of the inhabitants of these parts, who are thus afflicted, by the parasite in question, the presence of which induces a highly saline condition of the urine, and the formation of crystalline deposits around the ova.

The success which attends the treatment of intestinal parasites leads one to hope that some specific may be found against Bilharzia, which, long after the disappearance of the more obvious evidence of its presence—hæmaturia—is liable to prove a very dangerous associate. But the case of the urinary parasite is very different from that of the intestinal. The Bilharzia is not simply attached to the free surface of the mucous membrane, but lies within the orifices of the smaller veins; and the substances which pass through the kidney on their way out of the system are very few compared with those which find a more direct passage through

the intestinal canal. It appears, therefore, that in order to fulfil the first object, we must either sufficiently saturate the blood with a remedy poisonous to the parasite, or lead through the kidneys some substance which may prove hurtful to it. The aromatic diuretics (essential oils), such as turpentine, copaiba, cubebs, bucco, give some promise. Santonine, being an anthelmintic which becomes diffused in the blood, is worthy of a persevering trial. I have found that a draught, composed of ℥xv each of oil of turpentine and male fern, and ℥v of chloroform, in ℥ij of tragacanth mixture, given every morning, brought away great numbers of the ova. The saline condition of the urine is much diminished, and the renal irritation and pain due to the presence of crystalline concretions are much relieved by the administration of bicarbonate of potash in copious draughts of water. The alkali dissolves the uric acid, which I believe to be the cementing medium of the oxalic deposits, and thus the disintegration of the calculi is facilitated, and their formation prevented.

Here I must conclude. I have sought in vain for more knowledge of the complete sexual animal, but with this I feel sure that we shall soon become acquainted. I have made known the chief facts above recorded to Mr. Dunsterville, who has already communicated them to a medical friend at the Cape; and, as Mr. Dunsterville will himself shortly return thither, we shall not long, I feel convinced, remain ignorant of the whole of the history of that animal, the early stage of whose existence I have had the pleasure of bringing before you. I have been minute in my description of the forms, and particular in my illustration of them, because during the life of the patient they must needs constitute the chief, if not the only, evidence of the particular disease to which they give rise.

Appendix.

On February 26th, 1864, my friend Mr. Dunsterville wrote me as follows:—"You will be sorry to hear that my younger son passed a small calculus a day or two ago, similar to those passed by his brother—confirming your anticipations." This is an interesting fact, since it shows how soon after the disappearance of the hæmaturia another disease, apparently unassociated with it, but really dependent upon the same cause, may manifest itself. The young gentleman alluded to had not been troubled with hæmaturia for more than a year, and believed himself to be perfectly free from any urinary or renal disorder. His urine, as I have previously recorded, invariably contained ova of the parasite, and usually a slight deposit of oxalates, but not sufficient to excite his attention.

With regard to the distribution of the disease, I have since ascertained the following additional facts—viz., (1) that the disease prevails in Natal; (2) that in the Cape Colony it appears to be limited to Port Elizabeth and Uitenhage.

Dr. J. W. Johnston, resident for four years in Natal as assistant-surgeon to the 85th Regiment, has made the following communication to my friend Dr. E. Symes Thompson:—"Hæmaturia prevails to some extent among the children of the civil community of Natal, and attacks both sexes, but boys more frequently than girls. I could not determine satisfactorily the source of the hæmorrhage; sometimes the blood would be uniformly diffused in the urine; occasionally in the same patient, after the bladder was emptied, about a teaspoonful of blood would be passed, sometimes with, generally without, pain; at other times

coagulated blood would be passed, having the size and shape of the ureter. The hæmorrhage appeared to be passive, the result of simple congestion. There was neither lumbar pain, pyrexia, irritability of the bladder, turbid urine, or other indications of nephritis or vesical disease."

Adults are also liable to become affected. Mr. Joseph Henderson, whose acquaintance I have lately had the pleasure of making, and who has resided for many years in Natal, informs me that a family of colonists, consisting of three adult brothers, having settled between Port Natal and Pietermaritzburg, on the banks of the Sterk Spruit,[1] a tributary of the Umlazi, became affected with hæmaturia, and were obliged, after a residence of two years, to leave the country on this account. They believed the water of this stream to be the cause of the disease. Mr. Henderson writes me the following, which has an obvious bearing upon the identity of the disease prevalent in Mauritius:—"A friend of mine from the colony, who is now with me, says hæmaturia is a very common complaint in Natal, and names several people who were subject to it; he also says it is common in Mauritius."

As to the prevalence of the disease in the Cape Colony, Mr. George Saunders, staff-surgeon, has most obligingly furnished me with the result of his own observations, and also those of some of his friends. He writes—"While at Port Elizabeth I was greatly struck with the number of cases of hæmaturia in young boys, and on inquiry was informed that the disease was very common at Uitenhage, which is about ten miles distant from Port Elizabeth. I never met with hæmaturia at Graham's Town, which is ninety-five miles from Port Elizabeth, nor at Fort Beaufort or Alice, fifty miles up the country."

Mr. Robert Speedy, of the 45th Regiment, writes—

[1] The Sterk Spruit is a small stream about thirty miles from D'Urban, on the old road between D'Urban and Pietermaritzburg.

"Hæmaturia was not prevalent either at East London or King William's Town. I know that at Uitenhage many cases occurred. I met two gentlemen from the Cape who were labouring under the affection in an aggravated form. They stated that they had derived little benefit from treatment."

After reading the cases narrated by Chapotin, Salesse, and Rayer (op. cit., p. 56), I feel convinced that the disease prevalent in Mauritius is in all respects identical with that existent in parts of the neighbouring continent. The following extracts carry conviction with them, while they serve at the same time to indicate the theories which up to the present time have been held respecting the cause of endemic hæmaturia:—"In the Isle-of-France children of both sexes are affected from the tenderest age with hæmaturia, which announces the weakness of the mucous membrane of the kidneys. With some the disease is slight and continual; with others it returns at intervals with different degrees of force. It generally disappears at the age of puberty, but it is often prolonged beyond that time. The hæmaturia is frequently replaced by attacks of nephritic colic, which appears to depend as much upon a too abundant mucous secretion as upon engorgement of the blood-vessels of that part from the presence of renal calculi" (Chapotin). "Three fourths of the children of the Isle-of-France are attacked with hæmaturia. Masturbation and spiced dishes are the determining causes of it. The malady has also been attributed to a bad quality of the water employed for drink" (Salesse).

Prophylaxis.—As the prevention of the disease is a matter of the gravest importance to the communities amongst which the parasite is found, a few suggestions on this subject, for the consideration of those who are more immediately concerned, will not be out of place. According to the observations of Professor Siebold on the trematode worms, it may safely be assumed that between the ciliated embryo above described and the adult sexual animal there are probably two

other distinct forms, which serve to complete the chain of metamorphoses connecting these two extremes of development. What these forms are, and what their transmigrations, are the questions which require careful elucidation. The ciliated embryo is adapted for an aquatic existence. Swimming freely about, these minute organisms probably come in contact with certain mollusca, and become developed within them into what have been called *cercaria-sacs*, but which may perhaps be more significantly termed *free pseudovaria*. In the interior of these, we may still further assume, little caudate worms, or *cercariæ*, are budded off, and, leaving the pseudovarium, bore their way out of the body of the animal, to again become free inhabitants of the water, from whence they may be directly transferred to the alimentary canal of man, or indirectly by means of some other animal used by him as food. Once admitted to the body of a suitable vertebrate animal the cercaria loses its tail, and becomes developed into the perfect sexual form. Fresh-water mollusca and fish are probably the victims selected by the parasite during its development through these intermediate stages. How far these assumptions are correct is the subject matter of further investigation. Whatever may be the truth, the following precautions are positively indicated:—1. The water should be conveyed from its source to its destination in covered channels, so that the ova contained in the urinary, and probably the fæcal, products of those infested with the parasite may be prevented mixing with it. It is obviously desirable to ascertain whether any of the domestic animals are the means of thus disseminating the parasite. 2. Drinking-water should be filtered. 3. Salads which may entangle small mollusca containing parasites, and uncooked molluscs and fish (as smoked fish), should be carefully avoided.

EXPLANATION OF PLATES II AND IIa.

Fig. 1.—*a*. Filaments and lumps of mucus, containing ova, derived from one specimen of urine. Natural size.
b. Ditto ditto from another specimen.

„ 2.—A portion of *b*, fig. 1, magnified 50 diameters.

„ 3.—Impregnated ova—*a*, *a*, *c*, as they appear in the fresh urine; *b*, *b*, after maceration in carbolic-acid water or glycerine; *d*, spherical mass, probably the escaped embryo mass, which has assumed a spherical figure. × 100.

„ 4.—Embryo mass, escaped from the longitudinally dehisced egg-case. × 100.

„ 5.—Ovum, imbedded in mucus. × 200.

„ 6.—Ovum, in a more advanced stage. × 320.

„ 7.—Ovum, × 200, common appearance, showing spherules of the embryo mass.

„ 8, 9.—Ova, after maceration in water acidulated with hydrochloric acid. Fig. 8 shows longitudinal dehiscence of the egg-case, and liberation of fatty spherules and granules. × 320.

„ 10.—Ciliated embryo. × 320.

„ 11.—Ciliated integument, probably belonging to an adult Bilharzia. × 100.

„ 12.—Elongated and waved form of ovum. × 320.

„ 13.—Another form of ovum, with abrupt spine. × 320.

„ 14.—Compound sacculated tube, probably the intestinal canal of the parasite. × 50.

„ 15.—Ciliated embryo escaping from the egg-case. × 320.

„ 16.—Another form of ciliated embryo. × 320.

Fig. 1.

Fig. 2.

× 50.

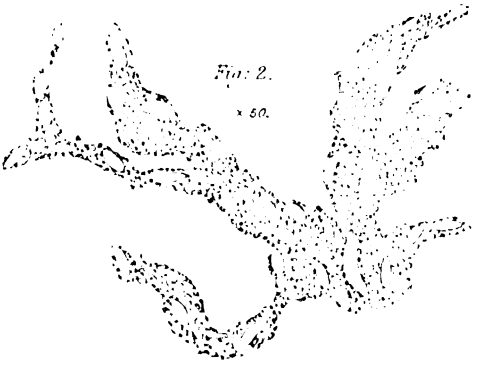


Fig. 4.

Fig. 3.

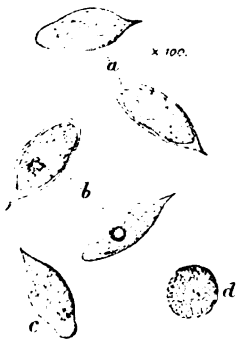


Fig. 5.

Fig. 6.

Fig. 7.

Fig. 8.

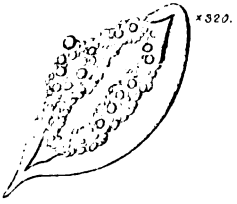


Fig. 9.

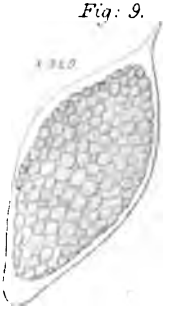


Fig: 10.

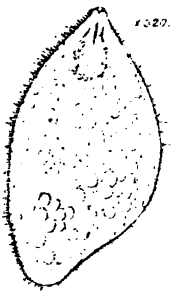


Fig: 11.

Fig: 12.

Fig: 13.

Fig: 14.

Fig: 15.

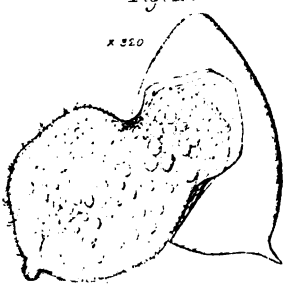
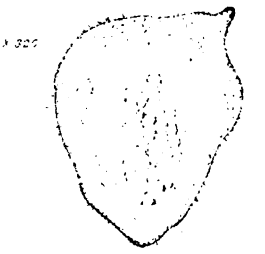


Fig: 16.



Dr John Harley, del ad nat. G.H.F

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SOME ACCOUNT

OF THE

AMPUTATIONS PERFORMED AT ST. BARTHOLOMEW'S HOSPITAL,

FROM THE 1ST JANUARY, 1858, TO THE 1ST OCTOBER, 1863.

BY

GEORGE WILLIAM CALLENDER, F.R.C.S.,

ASSISTANT-SURGEON TO ST. BARTHOLOMEW'S HOSPITAL.

Received Dec. 28th, 1863.—Read Feb. 9th, 1864.

THE following are some particulars of the amputations performed at St. Bartholomew's Hospital, the cases being so arranged, in a series of tables, as to show for a number of consecutive years the totals of deaths and of recoveries in male and female patients.

The operations comprise all the principal amputations, excluding, that is to say, those which involved the removal of parts only of the hands or feet; including, however, for the latter, amputations of whatever kind performed at the ankle-joint. Two amputations at the knee-joint (for disease) are reckoned with operations at the thigh.

The amputations placed first on the tables are those for injuries after various accidents, arranged under the usual headings of primary and of secondary operations.

To these succeed the amputations for disease and for malformation, and I confess to some hesitation before

deciding upon the plan I have adopted for their classification.

Some of these operations were unavoidably performed under critical circumstances; some, on the contrary, were postponed until the opportunity arrived which promised best for their after success. The latter group of cases, by far the more numerous one, embraces not merely amputations for distortion, for malformation, and for various tumours, but of necessity comprehends the greater number of amputations for articular disease—in fact, all those chronic maladies of the knee, ankle, or other joints, which do not threaten the life of the patient, but which, remaining stationary, or inclining to grow worse, invite removal of the diseased structures from the known hopelessness of regaining a useful condition of the limb. The shorter list of cases includes certain acute joint affections; some chronic articular diseases, ending with exhaustion of the patient, and threatening to kill; some cancer growths, with outstanding ulcers, enfeebling by their discharges or by their hæmorrhages; and such accidents as those which arise from the bursting of an aneurism into the tissues of a limb.

Had I been able, in a manner satisfactory to myself, to determine to which group each particular case by right belonged, or had I been able, after arranging these amputations as best I could, to show any material difference in their respective rates of mortality, I might have separated the operations for disease or for malformation into two classes—first, as amputations of expediency; secondly, as amputations of urgency. I have not done so, and I believe that for a statistical record the more practical, as it is the more simple, plan is to retain all these cases under the one heading given in the accompanying tables, and subsequently to point out a certain number which seem to require separate consideration.

I have thought it unnecessary to name the kind of operation selected for individual cases—that is to say, whether flap, circular, or other incisions were employed, or to allude to minor details, such as the use or the avoidance

of ligatures on veins, and the time and manner of first dressing the stumps; since I find that the influence, if any, thus exercised upon the general results, cannot be represented by figures.

It seems needless also to do more than recall the fact that, of amputations at any particular region of the body, those which are nearest to the trunk are, in themselves, least easily recovered from.

Whilst believing in the interest which attaches to these and to similar records, I do not assign much importance to their statistical evidence. It is well, however, that attention should be drawn to such facts as those contained in these tables, in order that we may from time to time compare the results of the amputations of the present day with the experience of the past, in the hope that in a lessened mortality we may be justified in assuming an improved surgery.

But to obtain data for accurate comparison, it is, I conceive, essential that our cases should be arranged so as to give in consecutive order, for periods however brief, a year by year account of these operations, and that the amputations in each set of cases should have been performed as far as possible under the same influences of locality and of ward conditions. And here I should state that the arrangements of the wards of the hospital have been continuously the same, in all important particulars, during the eleven years over which the following cases have been distributed.

TABLE I.

Principal Amputations performed during the Years—

		1853.				1854.				1855.				1856.				Totals.			
		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.	
		M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Primary Amputations.	Shoulder-joint ...	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—
	Arm	1	1	—	1	2	—	—	—	—	—	—	—	2	—	—	—	5	1	—	1
	Forearm	2	—	—	—	4	—	—	—	4	1	—	—	2	—	—	—	12	1	—	—
	Thigh	1	2	—	1	2	—	1	—	1	—	—	—	2	—	—	—	6	2	1	1
	Leg	—	—	—	—	4	—	—	—	—	—	—	—	3	—	1	—	7	—	1	—
Secondary Amputations.	Arm	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	1	—	1	—
	Fore-arm	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	—	—	—
	Thigh	4	—	1	—	1	—	1	—	1	—	—	—	—	—	—	—	6	—	2	—
	Leg	1	—	1	—	1	—	1	—	—	—	—	—	—	—	—	—	2	—	2	—
All other Amputations.	Shoulder-joint ..	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Arm	—	—	—	—	—	—	—	—	—	—	—	—	2	—	1	—	2	—	1	—
	Forearm	—	—	—	—	2	—	—	—	2	1	—	—	2	—	—	—	6	1	—	—
	Hip-joint	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
	Thigh	2	3	—	—	7	1	1	1	7	2	—	1	3	4	1	1	19	10	2	3
	Leg	6	1	2	—	5	1	3	—	2	3	—	—	7	2	—	1	20	7	5	1
	TOTALS.....	18	7	4	2	28	2	7	1	17	7	—	1	25	6	4	2	88	22	15	6

TABLE II.

Principal Amputations performed during the Years—

		1857.				1858.				1859.				1860.				Totals.			
		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.	
		M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Primary Amputations.	Shoulder-joint ...	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	1	—	—	—
	Arm	2	—	—	—	—	—	—	—	3	—	—	—	2	1	—	—	7	1	—	—
	Forearm	—	—	—	—	1	—	—	—	4	—	—	—	1	—	—	—	6	—	—	—
	Thigh	2	—	—	—	—	—	—	—	1	—	1	—	—	—	—	1	3	—	1	1
	Leg	2	—	—	—	1	—	—	1	3	—	—	1	1	—	—	1	7	—	—	3
Secondary Amputations.	Arm	—	—	—	—	—	—	1	—	—	—	—	—	1	—	—	—	1	—	1	—
	Forearm	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	1	—	—
	Thigh	—	—	—	—	—	—	—	—	1	—	—	—	1	—	—	—	2	—	—	—
	Leg	1	—	1	1	2	—	—	—	—	1	—	—	—	—	—	—	3	1	1	1
All other Amputations.	Shoulder-joint ...	1	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	1	—	—	1
	Arm	—	—	—	1	—	—	1	—	1	1	—	—	—	2	—	—	1	3	1	1
	Forearm	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—
	Hip-joint	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—
	Thigh	7	—	2	1	4	—	2	1	5	5	2	2	6	1	2	2	22	6	8	6
	Leg	3	—	2	—	4	2	1	—	2	1	—	—	6	1	—	—	15	4	3	—
	TOTALS.....	18	—	6	3	13	2	5	3	20	9	3	3	19	5	2	4	70	16	16	13

TABLE III.

Principal Amputations performed during the Years—

		1861.				1862.				1863.				Totals.				Gross Totals (Tables I, II and III).						
		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.		Totals Male and Female.	Per cent. of deaths.	Per cent. of deaths as corrected.
		M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.			
Primary Amputations.	Shoulder-joint ...	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—	2	—	—
	Arm	2	1	—	—	4	—	—	—	2	—	—	—	8	1	—	—	20	3	—	1	24	4·1	—
	Forearm	2	—	—	—	—	—	—	—	—	—	—	—	2	—	—	—	20	1	—	—	21	—	—
	Thigh	—	—	1	—	1	—	—	—	1	—	1	—	2	—	2	—	11	2	4	2	19	31·5	18·7
	Leg	1	—	2	—	—	—	1	—	3	1	1	—	4	1	4	—	18	1	5	3	27	29·6	20·8
Secondary Amputations.	Arm	—	—	—	—	1	—	—	—	1	—	1	—	2	—	1	—	4	—	3	—	7	42·8	—
	Forearm	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—	2	—	—
	Thigh	—	—	—	—	1	—	1	—	—	—	2	—	1	—	3	—	9	—	5	—	14	35·7	—
	Leg	1	—	—	—	—	—	—	—	2	—	1	—	3	—	1	—	8	1	4	1	14	35·7	—
All other Amputations.	Shoulder-joint...	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	1	2	—	—
	Arm	—	1	—	—	3	2	—	—	—	—	1	—	3	3	1	—	6	6	3	1	16	25·	20·
	Forearm	1	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	8	1	—	—	9	—	—
	Hip-joint	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	1	—	—
	Thigh	8	4	1	—	5	4	1	—	9	4	3	2	22	12	5	2	63	28	15	11	117	22·2	21·2
	Leg	7	4	2	1	6	1	1	—	3	2	1	—	16	7	4	1	51	18	12	2	83	16·8	16·
	TOTALS.....	22	10	6	1	21	7	4	—	21	7	11	2	64	24	21	3	222	62	52	22	358	20·6	19·3

TABLE IV.

Causes of Death in Seventy-four Cases.

	Complications.		Recurrent hæmorrhage.	Exhaustion.					Secondary hæmorrhage.	Consequent or aggravated disease of internal organs.	Totals.
	Independent injury.	Independent disease.		Within 24 hours.	At a later period.	With gangrene.	With erysipelas.	With pyæmia.			
AMPUTATIONS.											
Primary—											
Shoulder-joint	—	—	—	—	—	—	—	—	—	—	—
Arm	—	—	—	—	—	—	—	—	—	1	1
Forearm	—	—	—	—	—	—	—	—	—	—	—
Thigh	3	—	1	—	1	—	—	—	1	—	6
Leg	3	—	1	—	2	—	—	—	—	2	8
Secondary—											
Arm	1	—	—	—	—	—	—	1	1	—	3
Forearm	—	—	—	—	—	—	—	—	—	—	—
Thigh	—	—	—	—	2	—	—	1	1	1	5
Leg	—	—	—	—	—	3	—	—	1	1	5
All other—											
Shoulder-joint	—	—	—	1	—	—	—	—	—	—	1
Arm	—	—	—	1	—	1	—	2	—	—	4
Forearm	—	—	—	—	—	—	—	—	—	—	—
Hip-joint	—	—	1	—	—	—	—	—	—	—	1
Thigh	—	2	—	1	5	—	2	11	1	4	26
Leg	—	—	—	—	5	—	—	5	1	3	14
TOTALS	7	2	3	3	15	4	2	20	6	12	74

Primary Amputations.

Of 93 primary amputations, 78 recovered, 15 died. Of the 47 amputations performed at the upper extremity, 2 at the shoulder-joint and 23 at the arm recovered. Of the amputations at the forearm, 21 operations were recovered from. One amputation at the arm ended fatally. Of 19 amputations at the thigh, 13 recovered; 6 died, or in the proportion of 31·5 per cent.; but of the fatal cases, 3 died from injuries independent of the operation, so that to obtain the correct rate of mortality these ought to be deducted, as in each the fatal issue could in no way be associated with the amputation; the per-centage thus corrected is 18·7.

Of 27 amputations at the leg, 19 recovered; 8 died, or 29·6 per cent., or, deducting 3 cases of death from other injuries, 20·8 per cent.

Of the total number of amputations at the upper extremity, the per-centage of deaths is 2·1; of the lower extremity, 30·4, or, as corrected, 20; the per-centage of deaths for all primary amputations is 16·1, or, when corrected, 10·3.

Of 15 males who underwent amputation at the thigh, 4 died, giving a death-rate of 26·6 per cent.; of 23 who suffered amputation at the leg, 5 died, or 21·7 per cent.; but of 4 females operated upon, 2 died after amputation at the thigh, or 50 per cent.; and of 4, 3 died, or 75 per cent., after amputation at the leg.

The age in fatal cases (male and female) averaged 47 years. The age in cases of recovery (male and female) averaged 27 years.

Thus, 16·1 per cent. of all primary amputations, or 1 in 6·2, prove fatal, or, as corrected, 10·3, that is 1 in 9·6; and if the age of the fatal cases, which averages 47 years, be taken into consideration, I am justified in stating, for children and for adults under 40, that an unfavorable result after these amputations is an exceptional occurrence.

Secondary Amputations.

The secondary amputations number 37, and of these 24 recovered, 13 were fatal. Of amputations at the arm, the rate of mortality was 42·8 per cent.

Nine amputations at the thigh did well, 5 died, or 35·7 per cent.; of the leg, 35·7 per cent. proved fatal.

Thus, after amputations at the upper extremity, the mortality is at the rate of 33·3 per cent., and after those of the lower extremity 35·7 per cent.; whilst 35·1 per cent., or 1 in 2·8, of all secondary amputations, prove fatal.

Only 3 females figure in this division; 1 recovered after amputation at the forearm; of 2 amputations at the leg, 1 recovered, 1 (æt. 73) died.

Of male patients who underwent secondary amputation, 3 died; 42·8 per cent. after amputation at the arm, 35·7 per cent. after amputation at the thigh, and 33·3 per cent. after amputation at the leg.

The age of fatal cases (male and female) averaged 48 years. The age in cases of recovery (male and female) averaged 35 years.

Taking primary and secondary amputations together, 7·1 per cent. of those of the upper extremity and 32·4 of those of the lower extremity prove fatal, and 21·5 per cent., 1 in 4·6, of the total of traumatic amputations.

Amputations for Disease or for Malformation.

There are 228 amputations for disease or for malformation; 182 recovered; 46 died, or 20·1 per cent. After amputation at the shoulder, 1 recovered, 1 died; at the arm the mortality is at the rate of 25 per cent. All the amputations at the forearm recovered; 1 operation at the hip-joint terminated in death. Of 117 amputations at the thigh, the rate of mortality is 22·2 per cent.; of 83 cases of amputation at the leg, the death-rate is 16·8 per cent.

Of males, there died 33·3 per cent. after amputation at

the arm, 19·2 per cent. after amputation at the thigh, 19 per cent. after amputation at the leg. Of females, 14·2 per cent. died after amputation at the arm, 28·2 per cent. after amputation at the thigh, 10 per cent. after amputation at the leg.

Thus, of all these amputations, 20·1 per cent. proved fatal; but of those performed at the upper extremity, 18·5 per cent. died; whilst of those which involved the lower, 20·3 per cent. ended fatally.

The age in fatal cases (male and female) averaged 38 years. The age in cases of recovery (male and female) averaged 32 years.

Of the fatal operations, 2 at the arm were undertaken for erysipelas, 1 for gangrene, 1 for cancer; 14 at the thigh were needed for chronic and 3 for acute disease of the knee-joint, 1 for necrosis, 1 for secondary hæmorrhage after removal of an exostosis, 1 for gangrene, 1 for malformation, 4 for cancerous or other tumours, 1 for chronic ulceration; 8 amputations at the leg were for disease of the tarsus or of the ankle-joint or for malformation, 2 for gangrene (1 for senile gangrene), 1 for cancer, and 3 for chronic ulceration.

A certain number of cases—not many, it is true—must, however, be separated from this division of amputations—cases in which the operation was performed in consequence of mischief setting in after some surgical procedure, or after some slight injury, as, for instance, when gangrene or erysipelas has unexpectedly followed the original hurt, and rendered amputation necessary. Though allied to the secondary amputations by many of the conditions by which they are surrounded, these cases differ from them in the nature of the shock they have primarily endured, which has been a slight one if from a trifling hurt, or has been sustained under the protection of chloroform if from a surgical operation. Moreover, these cases have never passed through the stage in which the question of primary amputation could have been raised, and therefore, for purposes of

comparison, they are, I think, best considered apart. If it is thought desirable, it is an easy matter to add them to the list of secondary amputations.

I deduct on their account 2 amputations at the arm, 1 of which proved fatal from gangrene; 4 amputations at the thigh, 1 of which died from gangrene, and a second from exhaustion; and 2 at the leg, 1 proving fatal from extension of gangrene.

If these cases are subtracted from the totals of amputations for disease, the rates of mortality will remain—for the arm 20 per cent., for the thigh 21·2, and for the leg 16 per cent.

It follows that of the total 358 amputations the rates of mortality are as under:

Of all primary amputations, 16·1 per cent. (1 in 6·2), or, as corrected, 10·3 per cent. (1 in 9·6).
Of all secondary amputations, 35·1 per cent. (1 in 2·8).
Of all traumatic amputations, 21·5 per cent. (1 in 4·6), or, as corrected, 17·7 per cent. (1 in 5·6).
Of all amputations for disease or malformation, 20·1 per cent. (1 in 4·9).
Of all amputations at the upper extremity, 10·8 per cent.
Of all amputations at the lower extremity, 23·6 per cent.
Of all amputations, 20·6 per cent. (1 in 4·8), or, as corrected, 19·3 per cent. (1 in 5·1).

Thus, whilst the figures I have ventured to bring under the notice of the Society agree in some particulars with results heretofore recorded, they differ from them in others, and chiefly in the per-centage rates of mortality after primary and secondary amputations.

Causes of Death.

Influence of Age.

Old people are little able to resist the shock of the more serious amputations. Ten of the patients operated upon were over 65, and the following was the result in each case:

Male, æt. 72,	primary amputation	at the leg.	Died.
„ æt. 70,	„	„	„
Female, æt. 79	„	„	„
„ æt. 73,	secondary amputation	at the leg.	Died.
Male, æt. 66,	amputation	at the thigh for disease.	„
„ æt. 76,	„	leg „	„
„ æt. 67,	„	„ „	Recovered.
„ æt. 69,	„	„ „	„
Female, æt. 67,	„	arm „	„
„ æt. 76,	„	„ „	„

Here the lesser amputations for disease offer the more favorable results, as might, indeed, have been anticipated. The influence of age, however, is best shown by comparing the averages in fatal and in successful cases. The contrast is most marked in primary amputations, and least so in amputations for disease.

Amputations.	Average age in fatal cases.	Average age in cases ending favorably.
Primary . . .	47 .	. 27
Secondary . . .	48 .	. 35
For disease . .	38 .	. 32

Influence of Sex.

After primary amputations the mortality is much greater for females than it is for males, the death-rate standing at 62·5 per cent. for the former, at 23·6 for the latter sex.

After amputations at the thigh for disease, or for malformation, the deaths are 19·2 per cent. for males, but 28·2 for females. After amputations at the leg only 10 per cent. for females, but 19 per cent. for males.

Thus, although females recover, if not more easily than, certainly as readily as, males, from the shock of certain amputations, they do not rally so easily after the severe shocks which precede and accompany primary amputations, nor after the depression consequent upon amputation at the thigh.

The rate of mortality on the totals of cases is 18·9 per cent. for males, but 26·1 per cent. for females.

TABLE—*Showing the Totals of Deaths and of Recoveries in 227 Cases, as influenced by the Age and Sex of the Patients, January, 1857, to October, 1863.*

Age.....	1—10				10—20				20—30				30—40				40—50				50—60				60—70				70—80			
	Recovered.		Died.		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.		Recovered.		Died.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
Amputations at the																																
Upper Extremity	...	1	...	...	16	...	1	...	4	2	2	1	4	1	...	...	7	...	1	1	3	3	...	...	...	1	...	...	...	1	...	. .
Thigh	3	1	...	...	14	7	1	1	16	6	3	4	7	2	3	2	8	2	8	1	4	...	3	1	...	...	2	...	...	...	...	...
Leg	3	...	...	...	9	4	...	...	13	5	3	...	9	3	2	...	6	...	...	2	5	1	5	...	3	...	...	1	...	...	3	2
	6	2	...	...	39	11	2	1	33	13	8	5	20	6	5	2	21	2	9	4	12	4	8	1	3	1	2	1	...	1	3	2
	8				53				59				33				36				25				7				6			
					1 in 17·6				1 in 4·5				1 in 4·7				1 in 2·7				1 in 2·7				1 in 2·3							

CAUSES OF DEATH—*continued.*

Primary Amputations.

The fatal case amongst primary amputations at the upper extremity was that of a female, æt. 56, who died from tetanus on the eleventh day.

Amongst operations at the lower extremity, the thigh amputations proved fatal—(1) to a male, æt. 50, from shock and other injuries (suicidal), at the eighth hour; (2) to a female, æt. 27, from shock and other injuries (suicidal), at the ninth hour; (3) to a male, æt. 17, from fractured ribs and pleurisy, on the twelfth day. These are the three cases deducted for the corrections previously referred to. (4) To a male, æt. 41, from shock and recurrent hæmorrhage, at the twelfth hour; (5) to a female, æt. 38, from exhaustion, on the ninth day; (6) to a male, æt. 28, from secondary hæmorrhage, on the tenth day.

Amputations at the leg proved fatal—(1) to a male, æt. 31, from fracture of the pelvis and other injuries, at the twelfth hour; (2) to a male, æt. 72, from fracture of the opposite leg and other injuries, on the fourteenth day; (3) to a female, æt. 78, from other injuries, from the shock of which she never rallied, on the fourth day; and these are the three cases referred to as deducted in correcting the per-centage of deaths; (4) to a male, æt. 47, from exhaustion, on the third day; (5) to a male, æt. 53, from delirium and exhaustion, on the seventh day; (6) to a female, æt. 48, from exhaustion, on the twenty-fifth day; (7) to a female, æt. 60, from lung congestion and exhaustion, on the fourth day; (8) to a male, æt. 70, from recurrent hæmorrhage, on the second day.

Thus, of 15 fatal cases, 6 died from traumatic complications, 2 from recurrent hæmorrhage, 3 from exhaustion at some time after the first twenty-four hours, 1 from tetanus, 1 from secondary hæmorrhage, 1 from delirium tremens, and 1 from congestion of the lungs.

So that traumatic complications proved fatal at the rate of 40 per cent., and exhaustion at the rate of 20 per cent., of the total number of deaths.

Secondary Amputations.

The following was the cause of death after the several secondary amputations:

Of amputations at the arm—(1) a male, æt. 29, died from other injuries on the third day; (2) a male, æt. 46, died from pyæmia on the sixteenth day; (3) a male, æt. 40, sank from purpura and secondary hæmorrhage (blood-oozing) on the twelfth day.

Of amputations at the thigh—(1) a male, æt. 48, died from exhaustion on the eighth day; (2) a male, æt. 52, died from secondary hæmorrhage on the 11th day; (3) a male, æt. 47, sank from pyæmia on the ninth day; (4) a male, æt. 50, died from secondary hæmorrhage, but chiefly from diarrhœa, on the twelfth day; (5) a male, æt. 42, sank from exhaustion and syncope on the tenth day.

Amputations at the leg proved fatal—(1) to a male, æt. 54, from exhaustion and gangrene, on the thirteenth day; (2) to a male, æt. 28, from secondary hæmorrhage, on the fifth day; (3) to a female, æt. 73, from exhaustion and gangrene, on the fourth day; (4) to a male, æt. 64, from exhaustion and gangrene, on the fifth day; (5) to a male, æt. 50, a half-witted fellow, from fits and exhaustion, on the fifteenth day.

Of 13 fatal cases, 5 sank from exhaustion, 3 from secondary hæmorrhage, 1 died from traumatic complications, 2 from pyæmia, 1 from diarrhœa, and 1 from cerebral disorder.

So that exhaustion was again the chief cause of death, 38·4 per cent. sinking in this way. 23 per cent. died from secondary hæmorrhage, though in one of the cases under this heading the bleeding was a general oozing, associated with symptoms indicative of purpura. The number of fatal cases in this division is, however, so small that these percentages are of little value.

Of the total of traumatic amputations ending fatally it

may be noted that 28·5 per cent. sank from exhaustion, 25 per cent. from traumatic complications, 21·4 per cent. from hæmorrhage, and 7·1 per cent. from pyæmia.

Amputations for Disease or Malformation.

The cause of death in each fatal amputation at the upper extremity was as follows:

After amputation at the shoulder-joint, a female, æt. 21, sank from exhaustion and shock within twenty-four hours; after amputation at the arm, (1) a male, æt. 34, died from pyæmia on the seventh day; (2) a female, æt. 44, sank from exhaustion within twenty-four hours after amputation for erysipelas; (3) a male, æt. 26, died from pyæmia on the fifteenth day; and (4) a male, æt. 12, died from extension of gangrene within twelve hours.

Of 26 amputations at the thigh, pyæmia was the cause of death in 11 cases; independent disease in 2; exhaustion within twenty-four hours proved fatal in 1 instance, exhaustion at a later period was the cause of death in 5 cases; in 2 erysipelas proved fatal, in 1 secondary hæmorrhage, and in 4 various visceral complications.

Of 14 amputations at the leg, 5 died from pyæmia, 5 from exhaustion, 1 from secondary hæmorrhage, and 3 from visceral complications.

Amongst 46 fatal cases, exhaustion was the cause of death in 13, erysipelas in 2, pyæmia in 18, secondary hæmorrhage in 2, gangrene in 1. The remainder died, 2 from independent disease, 7 from visceral complications, 1 after amputation at the hip-joint, from recurrent hæmorrhage.

Thus, exhaustion was fatal in 28·2 per cent. of these cases, pyæmia in 39·1 per cent., and visceral complications in 15·2 per cent. For amputations at the thigh, exhaustion was fatal at the rate of 23 per cent., pyæmia at the rate of 42·3 per cent., and for amputations at the leg, exhaustion stands at 35·7 per cent., pyæmia at 35·7.

Taking the four chief causes of death after all amputa-

tions, we obtain the following rates of mortality in the totals of fatal cases:

Amputations.	Hæmorrhage. Per cent.	Pyæmia. Per cent.	Exhaustion. Per cent.	Visceral complications. Per cent.
Primary	20·	—	20·	6·6
Secondary	23·	15·3	38·4	15·3
Traumatic (all)	21·4	7·1	28·5	10·7
For disease	4·3	39·1	28·2	15·2

Of the total 74 fatal cases, 24·3 per cent. died from exhaustion, 27 per cent. from pyæmia, 12·1 per cent. from hæmorrhage, 16·2 per cent. from visceral complications.

After the operations performed for disease or for malformation, pyæmia proved fatal in 1 case of amputation at the arm for erysipelas and diseased elbow-joint; in 9 cases of amputation at the thigh, 2 for acute, and 7 for chronic disease, involving the knee-joint, and in 2 for malignant tumours; in 5 cases of amputation at the leg, 4 for tarsal or ankle-joint disease, including 1 case of malformation, 1 for chronic ulceration.

Exhaustion was the cause of death after amputation at the arm in 2 cases, once within twenty-four hours; after amputation at the thigh, for disease involving the knee-joint in 4 cases, for chronic ulceration in 1 case; after amputation at the leg, for disease of the ankle-joint in 1 case, for disease of the tarsus in 2 cases, for senile gangrene in 1 case, for chronic ulceration in 1 case.

Fatal erysipelas in 2 instances occurred after amputation at the thigh for chronic disease of the knee-joint (in 1 case combined with necrosis), proving fatal on the eighth and eleventh days. Both patients were females.

In the table (IV) showing the immediate cause of death in 74 fatal cases, 2 patients are stated to have died from independent disease. One, a female, had been an inmate of a lunatic asylum before her admission into the hospital, and sank from exhaustion on the fourteenth day, in consequence of an attack of violent mania, which commenced shortly after the operation. The other, a male, lived twenty-four days

after amputation at the thigh, and then died from cancer of the liver.

Three cases proved fatal from recurrent hæmorrhage; a male, æt. 47, died at the fifth hour, partly also from shock, after amputation at the hip-joint, for cancer involving the femur; another male died in the same way after primary amputation at the thigh, and a third male sank on the second day after primary amputation at the upper third of the leg. The last two cases were also much depressed by repeated administrations of chloroform.

Of the deaths from gangrene, a boy, æt. 12, died from the rapid extension of a gangrenous process which had set in prior to the operation, and which killed him within twelve hours.

The small number of deaths from shock within twenty-four hours is remarkable. Of the 3 cases tabulated as independent of traumatic complications or of recurrent hæmorrhage, the amputation at the thigh was of necessity performed on a male, æt. 36, when on the verge of death, in consequence of repeated secondary hæmorrhages and other mischief, after the removal of an exostosis from the thigh. A female, æt. 21, sank from shock after amputation at the shoulder; and another female, æt. 44, died within twenty-four hours after amputation above the elbow, but she had been previously reduced to death's door by erysipelas of the hand and arm.

The deaths ranged under the head "pyæmia" are cases of exhaustion complicated with some form of blood poisoning; they include also the cases of embolism and of clot softening (thromballosis).

Twelve patients died from disease of internal organs, consequent upon or aggravated by the operation. A female, æt. 56, sank from tetanus eleven days after primary amputation at the arm; a male, æt. 50, died of delirium tremens seven days after primary amputation at the leg; and a female, æt. 60, died from congestion of the lungs on the fourth day, also after primary amputation at the leg.

After secondary amputation at the thigh, a male, æt. 50,

sank from diarrhœa on the twelfth day; and after secondary amputation at the leg, another male, æt. 50, had a succession of fits, and so became exhausted on the fifteenth day.

Of the amputations for disease, a male, æt. 33, sank from choleraic diarrhœa on the fourteenth day; a female, æt. 40, from diarrhœa, on the seventh day; a female, æt. 21, from congestion of the lungs and phthisis, on the eighteenth day; a female, æt. 22, from phthisis, on the thirtieth day. All four after amputation at the thigh.

A male, æt. 20, subject to fits, having in one of his attacks contrived to burn his feet, and having in consequence to undergo, some time after, amputation at the leg, died with cerebral symptoms, somewhat suddenly, on the twelfth day. A male, æt. 54, died from restless delirium and phthisis on the seventh day, and another male, æt. 55, sank from pneumonia of the right lung on the twenty-sixth day. In both cases after amputation at the leg.

Fatal Days.

The following table shows the days on which 74 cases terminated fatally, and also the cause of death.

Days.	Causes of Death.	Totals.
1	6 from shock, exhaustion, and other injuries; 1 from gangrene, 2 from recurrent hæmorrhage	9
2	1 from recurrent hæmorrhage	1
3	2 from exhaustion	2
4	5 from exhaustion, 1 congestion of lungs	6
5	1 from exhaustion, 1 secondary hæmorrhage	2
6	1 from erysipelas	1
7	1 from exhaustion, 1 pyæmia, 1 secondary hæmorrhage, 1 from diarrhœa, 2 delirium	6
8	1 from exhaustion	1
9	1 from exhaustion, 3 pyæmia	4
10	2 from exhaustion, 2 pyæmia, 1 secondary hæmorrhage	5
11	2 from pyæmia, 1 tetanus, 1 erysipelas, 1 secondary hæmorrhage	5

Days.	Causes of Death.	Totals.
12 . .	1 from pyæmia, 3 exhaustion, 1 diarrhœa, 1 epilepsy, 2 secondary hæmorrhage .	8
13 . .	1 from exhaustion	1
14 . .	1 from pyæmia, 1 mania, 1 diarrhœa, 1 exhaustion	4
15 . .	4 from pyæmia, 1 fits . . .	5
16 . .	1 from pyæmia, 1 exhaustion . .	2
17 . .	1 from exhaustion	1
18 . .	4 from pyæmia, 1 congestion of lungs .	5
24 . .	1 from pyæmia, 1 exhaustion, 1 cancer of liver	3
25 to 35 .	1 from exhaustion, 1 phthisis, 1 pneumonia .	3
		74

Deaths from shock or from other injuries, or from both combined, take place within the first twenty-four hours; and within forty-eight hours the deaths from recurrent hæmorrhage occur. Exhaustion is most fatal about the fourth day. Secondary hæmorrhage is a cause of death from the fifth to the twelfth day; pyæmia from the seventh to the twenty-fourth, proving most fatal on the ninth, tenth, eleventh, fourteenth, fifteenth, and eighteenth days.

The days on which the deaths occurred in individual cases have been already given for primary and for secondary amputations. The following are the days on which exhaustion and pyæmia respectively proved fatal after amputations for disease or for malformation:

	Exhaustion.			Pyæmia.		
	Arm.	Thigh.	Leg.	Arm.	Thigh.	Leg.
Fatal day in each case	1st	4th	4th	15th	—	—
	1st	4th	10th	7th	9th	10th
	—	7th	12th	—	9th	14th
	—	12th	16th	—	10th	15th
	—	17th	24th	—	11th	18th
	—	36th	—	—	11th	—
				—	12th	18th
				—	15th	—
				—	15th	—
				—	18th	—
				—	18th	—
				—	24th	—

Cases of amputation, the patients remaining in the hospital until they died, not tabulated with the fatal cases, as the death in each instance was not accelerated by the operation.

1. A female, æt. 23, died after amputation at the thigh, from phthisis, on the 109th day after the operation. 2. A male, æt. 45, died from long-standing granular degeneration of the kidneys on the 102nd day after amputation at the thigh. 3. A male, æt. 24, died of phthisis ninety-three days after amputation at the thigh.

I may mention, as curious facts, that in the year 1854 a male recovered after primary amputation of both legs; that in one year, 1857, all amputations performed on female patients proved fatal, whilst in another year, 1862, all were recovered from. That in the year 1855 17 males suffered amputation, and all did well; that during the same year, in 25 amputations, 12 of which were performed at the thigh, there was only 1 death. In the year 1861 there were 24 consecutive operations, and but 1 fatal case; but in the year 1863, to the end of September, there were 13 deaths to 28 recoveries. Indeed, I add the cases occurring during the nine months of 1863 to those for the ten preceding years, on purpose to show how necessary it is to mass together a considerable number of consecutive operations before we have a chance of arriving at tolerably just conclusions. Had I, as was my first intention, omitted these nine months, the percentage mortality on the totals of cases would have stood, for the primary amputations, 15·4 instead of 16·1; for the secondary, 30 instead of 35·1; for amputations for disease, 19·2 instead of 20·1; and for the amputations of all kinds, 19·2 instead 20·6.

In 1858 and in 1863 I find respectively 5 and 8 consecutively fatal cases, but the deaths are due to a variety of causes, and not to any one more signally than the rest.

A CASE

OF

STRANGULATED FEMORAL RUPTURE,

WHERE, ON A FORMER OCCASION, THE NECK HAD BEEN TORN FROM THE BODY OF THE SAC, IN THE TAXIS, AND THE ESCAPING BOWEL HAD FORMED A SUBPERITONEAL POUCH, WHICH FINALLY ATTAINED AN EXTRAORDINARY SIZE.

BY

J. W. HULKE, F.R.C.S.,

ASSISTANT-SURGEON TO THE MIDDLESEX AND ROYAL LONDON OPHTHALMIC HOSPITALS.

Received Jan. 1st.—Read Feb. 9th, 1864.

In strangulated femoral hernia true reduction in mass has been proved by many well-authenticated cases to be a not very infrequent event, whilst tearing of the neck of the sac, with extrusion of the strangulated bowel between the peritoneum and abdominal muscles (which Mr. Birkett has shown to occur frequently in cases of inguinal hernia thought to have been reduced in mass[1]), is so rare an occurrence, that it is not described in Scarpa and Lawrence on hernia, nor in the text-books of surgery in common use. The loose manner in which the sac is commonly connected to the surrounding structures in femoral hernia so conduces

[1] 'Med.-Chir. Trans.,' vol. xlii, p. 247.

to true reduction in mass, that these connections are stretched or broken, so as generally to allow the sac to be displaced by a less force than is requisite to tear its neck; but the possibility of laceration of the neck of the sac in femoral hernia is shown by the following case.

CASE.—A woman, æt. 70, was admitted into the Middlesex Hospital with a strangulated femoral rupture, Tuesday night, October 12th. It had existed thirty years, had been several times strangulated, and always reduced by the taxis. The present strangulation took place on the preceding Sunday afternoon, during an attack of diarrhœa. The house-surgeon, who had reduced the rupture by the taxis in a warm bath when it was last strangulated, three months before, could not do so this time, and sent for me in the absence of the surgeon of the week, Mr. De Morgan.

She had a tender, tense, elastic, globular swelling in the left groin, of the size of a billiard-ball, circumscribable everywhere, except at its lower border, where it dipped into the femoral canal; retched frequently, was very exhausted, and her bowels had last acted the previous day.

Mr. De Morgan arriving, requested me to take charge of the patient, and gave me the advantage of his assistance.

Complete muscular relaxation having been induced with chloroform, I repeated the taxis for a few minutes unsuccessfully, and, supposing the case to be an ordinary femoral rupture, proceeded to operate by the customary incision along the inner border of the swelling.

After dividing the subcutaneous fatty tissue, and turning aside a dark-red, deeply congested, lymphatic gland, covered by a thin fascia, the sac was laid bare, and the deep femoral arch, Gimbernat's ligament, and part of the conjoined tendon, having been divided, an attempt was made to return its contents without opening it. As this was impossible, the sac was opened. It had all the usual characters of a common peritoneal sac, and contained a knuckle of dark, claret-coloured, small intestine, coated with lymph, and a few drachms of serum. The neck of the sac and some tight

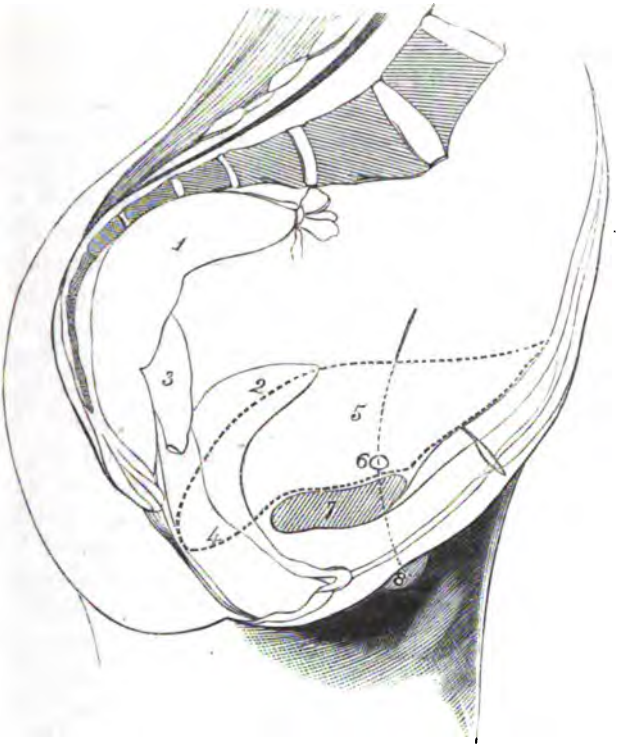
threads immediately outside it were next divided; but still the protruding bowel could not be returned into the belly, notwithstanding that so much space had been gained that the finger passed freely through the femoral ring. As the bowel was not adherent to the neck or body of the sac, the obstacle to its return was presumed to be inside the belly above the femoral ring, and in order to reach it Poupart's ligament was cut. Having gained considerably more space by this, on running the finger along the mesentery some deep transverse bands were felt and divided; but even after this the bowel could not be fairly replaced in the belly, but remained in the neck of the sac. Passed through the femoral ring into the belly, the finger could be moved freely in a large space, the anterior boundary of which, above and behind the pubis, felt like omentum; whilst directed towards the iliac fossa it met a rounded edge, such as a twisted mesentery might form, and beyond this, at its full depth, sometimes, but not always, the smooth inner surface of the peritoneum was felt. The exact nature of the obstacle could not be made out, and, as it was evident from the subsidence of the distension and congestion of the bowel that the strangulation was removed, the exploration was discontinued, and the wound closed with wire sutures.

13th, at 9 a.m., the lower part of the belly was very painful and tender. During the day the pain and tenderness increased, and she grew weaker, and died at 7 p.m., nearly seventeen hours after the operation.

At the examination of the body the knuckle of bowel was found where it had been left, in the bottom of the wound, and the finger could be passed easily through the femoral ring, as at the operation. On opening the belly by a crucial incision, and turning back the flaps, a large pouch was found outside the peritoneum, between it and the abdominal and pelvic walls. It reached upwards about three inches above the pubis, outwards as far as the deep epigastric artery on each side, downwards in front of the bladder, behind the pubis, at each side of the vagina, to the floor of the pelvis, and extended into the left sciatic notch. The

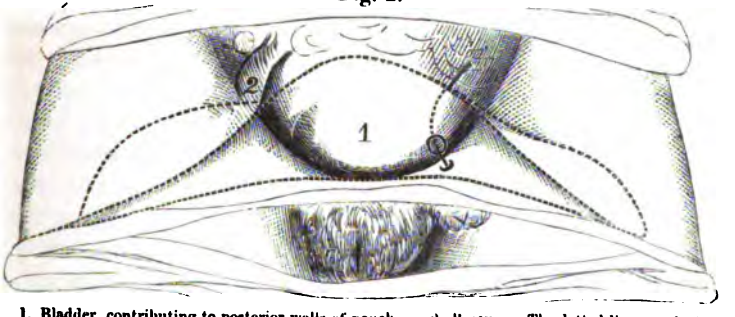
posterior wall of this immense pouch consisted of peritoneum, strengthened above by the fascia transversalis, and contained below the bladder, which had been displaced from behind the pubis towards the sacrum in the hollow of which the uterus was lying in the mesial line, at the left of the rectum, which descended on the right side. In this wall, opposite the left femoral ring, but about two and a half inches distant from it, there was a circular aperture, three quarters of an inch in diameter, by which the pouch communicated with the peritoneal cavity. It was evidently the original neck of the hernial sac. The margin was formed by smooth, opaque, white, thickened peritoneum, which extended a quarter of an inch upon the inner surface of the pouch, where it ended by a sharply defined cicatricial edge. Springing from the posterior wall, at the lower half of this edge, a slip of fascia ran forwards across the pouch, and lost itself in the fascia propria, in the femoral canal, behind the hernial sac. Through the aperture eight feet of small intestine had passed from the peritoneal cavity into the pouch; and it was also through it that the finger, directed towards the iliac fossa, had reached the smooth surface of the peritoneum at the operation. The inner surface of the pouch was smoothly lined with condensed cellular tissue, except in the neighbourhood of the left femoral ring, where there were several bands, some of which had been divided by the hernia knife. The knuckle of bowel in the sac, which had been strangulated, was continuous with that in the pouch. The entire bowel in the pouch was inflamed, and the inflammation had been most intense nearest the strangulated portion. The aperture in the posterior wall of the pouch did not exert any constriction.

Fig. 1.



1. Rectum, descending at the right side. 2. The bladder. 3. Uterus, thrown back into hollow of sacrum. 4. Elongated vagina. 5. Subperitoneal hernial pouch. The dotted line shows its limits. 6. Communication between this pouch and the peritoneal cavity; original neck of hernial sac. 7. Pubes. 8. Portion of hernial tumour in groin.

Fig. 2.



1. Bladder, contributing to posterior walls of pouch. 2. Rectum. The dotted lines mark the limits of the hernial pouch.

I conclude the following to be the true explanation :—On a remote occasion the neck had been completely torn by the taxis from the body of the hernial sac, a true reduction *en masse* being prevented by the smallness of the femoral ring, which in this case was very marked. The fascia propria had been torn at the same time, and the bowel squeezed up out of the body of the sac through the femoral ring, and, still girt by the neck of the sac, had insinuated itself under the peritoneum and fascia transversalis, near the femoral ring, where it formed a subperitoneal pouch. The strangulation ceased when the bowel re-entered the femoral ring, and was not caused by the neck of the sac. The pouch had been gradually enlarged by the entrance of fresh bowel from the peritoneal cavity, during which the neck of the sac became stretched, which made the entrance of additional bowel easier, and was removed further from the femoral ring.

The femoral ring, not the old neck of the sac, was the seat of strangulation on the present occasion. Death was caused by inflammation of the large extent of bowel in the subperitoneal pouch, which proceeded from the strangulated knuckle in the groin. The distension of the pouch to its utmost limits, so that it was unable to receive more in addition to the eight feet of bowel it already contained, would seem to have been the cause which prevented the return of the bowel from the sac after the free division of the structures at the inner side of the femoral ring. The survival of the patient after the reduction of the bowel, still girt by the neck of the sac, and the large dimensions which the subperitoneal pouch ultimately attained, are very remarkable.

ON THE

ABSORPTION OF DEAD BONE.

BY

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Can dead bone be absorbed? One is almost tired of the question. Still, it must be asked until it can be answered. Notwithstanding all that has been done on the subject the reply remains doubtful, for while, on the one hand, the most careful and accurate experiments, such as those of Mr. Gulliver, recorded in the twenty-first volume of the Medico-Chirurgical Transactions, have furnished only negative results; on the other hand, there are unquestionable facts, such as the absorption of ivory pegs when driven into bone, which compel us to admit the possibility of the occurrence.

In reflecting upon this evidence, thus apparently contradictory, it seemed to me that one all-important consideration had been neglected in the inquiry—the influence of *pressure* in determining the result. Thus, in the experiments which have been performed on the subject, and which have naturally led to the conclusion that dead bone may be kept amidst living tissues for weeks or months without losing the merest fraction of its weight, in these experiments the dead bone was kept in simple contact only with living parts; it appears that no considerable pressure was

maintained, whereas when ivory pegs are driven into bone extreme pressure is, of course, produced. In order to test this view, the following experiments were performed.

Experiment 1.

The outer surface of the right tibia of a young ass having been exposed by a short incision, a hole was drilled into it, and into this a peg, made out of the compact substance of the shaft of a human femur,[1] was very loosely inserted. The incision was then carefully and completely closed by silver sutures.

The corresponding part of the left tibia of the same animal having been exposed in like manner, a similar hole was drilled into it, and into this a peg, made of the same bone, was tightly hammered. The incision was then closed by silver sutures.

Eighteen days afterwards the pegs were examined. The wounds had suppurated, and were partially closed. The peg which had been loosely placed in the right tibia was in no way altered; its whole surface was still perfectly smooth. The peg which had been tightly hammered into the left tibia was visibly affected. The surface was roughened from the removal of very thin layers of bone in irregular patches.

After death the bones which had been operated on were found to be acutely inflamed. Upon section, the medullary membrane and cancellous tissue were of a bright-scarlet colour; even the compact texture was of a pinkish hue. In one case the greater portion of the medullary membrane was in a sloughing state.

Experiment 2.

The outer surface of the femur of a young rabbit having been exposed by a simple incision over it, a hole was drilled transversely through the shaft, and into this a conical peg

[1] In all these experiments the pegs and plates employed were made out of the compact substance of the shaft of a human femur.

was passed as far as it would go, its larger end being firmly impacted, while in the rest of its extent it lay comparatively free. The wound was closed by silver sutures, and soon completely healed.

At the end of six weeks the rabbit was killed. The peg was found exactly as it had been placed. All traces of disturbance in the surrounding parts had quite passed away. When the peg was extracted it was visibly corroded at the part which had been subjected to pressure.

Experiment 3.

The right femur of a young rabbit having been exposed, a small plate of bone was loosely attached to it by thin copper wire passed around them. The wound was then closed.

To the left femur a similar plate of bone was firmly secured by copper wire passed around them, and twisted up so tightly as to press the opposed surfaces forcibly together. The wound was then closed.

Both wounds suppurated for some time, and at last healed. The rabbit continued to be quite healthy, and increased rapidly in weight. At the end of six weeks it was killed.

When the skin was reflected, each plate was found imbedded in a mass of inspissated pus, which was invested by a thin, transparent, but well-defined, capsule. The plate which was loosely connected was not perceptibly altered, but the surface of the plate which was firmly pressed against the bone was in part corroded.

Experiment 4.

The front surface, towards its lower end, of one femur of a healthy young rabbit was exposed by a simple incision, and a hole was drilled through it. Through this a peg, which weighed eight grains, was hammered so as to transfix the bone.

A similar operation was then performed on the other femur, with this exception, that the peg, which also weighed eight grains, and was tightly hammered in, did not pass completely through the bone.

Both wounds were thoroughly closed by a continuous silver suture, and soon completely healed.

Five weeks and two days afterwards the rabbit, which did not appear to suffer materially from the operation, died. During the last fortnight of its life it had gradually lost flesh.

The external wound had been long closed. A large abscess was found around each peg in front of the femur, consisting of inspissated pus in a distinct and well-formed cyst. The pegs, which had been driven in, were firmly implanted. It required considerable force to draw them out.

The portion of each in contact with the bone had been obviously acted on. The surface was much roughened from removal of the superficial portion by absorption. The peg which had transfixed the bone had lost a grain of its weight, and the other almost as much.

Experiment 5.

The outer surface, towards the lower end, of the left femur of a healthy young rabbit was exposed by a simple incision, and a hole was drilled completely through it. Through this a conical peg, which weighed twelve grains, was tightly hammered so as to transfix the bone and project about the sixth of an inch beyond.

The front surface of the right femur, towards its lower extremity, was then exposed, and a thin plate of bone, weighing five grains, was secured in firm contact with it by means of copper wire passed around the femur, the ends of which were tightly twisted together over the plate. To increase the pressure, a conical peg of bone, weighing four grains, was afterwards pushed under the wire.

Both wounds were thoroughly closed by a continuous silver suture, and soon completely healed.

Seven weeks and three days afterwards the rabbit died. For the first six weeks it appeared to be well and lively, then it gradually lost flesh and strength.

Both external wounds were completely healed. An enormous abscess, in a distinct sac, was found around the peg in the left leg. A smaller abscess invested the plate in the right limb.

The peg in the left femur was firmly implanted, and it required some force to extract it. The portion which occupied the hole in the bone was obviously affected. The surface was much roughened from loss of substance. The portions projecting beyond were in no way altered. To one part of the peg a small thin plate of bone adhered very firmly. This was a portion of the posterior wall of the femur which formed the boundary of the hole. The connection between the fragment of bone and the peg must have been very intimate, for when the peg was extracted they not only came away together, but afterwards they resisted considerable force which was applied to separate them.

The plate and peg were found tightly bound to the other femur by the copper wire, which now appeared to pass beneath the periosteum, as if this membrane had grown over it. Around the edges of the plate some new bone had been, as it were, heaped up. When the plate and peg were removed, their surfaces were still quite smooth. They did not appear to have been affected in any way.

It is very difficult in this way, or in any other that I could devise, to maintain any considerable amount of pressure, or pressure at all corresponding to that which is obtained by driving a peg into bone.

Experiment 6.

The front surface, near the lower end, of the left femur of a healthy young rabbit was exposed, and a hole was drilled through it. Into this a peg, which weighed four

grains, was thrust. It accidentally produced fracture of the femur across the hole. The peg was left lying loosely between the ends of the fractured bone, and the wound was closed by a continuous silver suture.

A similar hole was made in the corresponding part of the right femur, and a similar peg, of equal weight, was tightly hammered in. The wound was then closed in like manner.

The rabbit continued apparently well for some time. Then around either femur a large collection of pus formed, and eight weeks and four days after the operation the rabbit died.

Both external wounds had been long before death completely healed.

There was an enormous collection of pus around the left femur, and in the wall of the cyst which contained it the peg was found imbedded, having evidently been carried out away from the bone. It had undergone no change of any kind. There was no repair of the fractured bone.

A much smaller collection of pus was around the right femur. The peg was found loose in the hole, for this had been drilled at the junction of the epiphysis with the shaft of the bone, and the peg, driven in, had caused their separation. Thus it lay loosely between them—there was no reunion—and had not undergone any change whatever.

Experiment 7.

A hole was drilled through the lower part of the shaft of the left femur of a healthy young rabbit, and into this a peg, which weighed four grains, was loosely inserted. The wound was closed by a continuous silver suture, and soon healed throughout.

A similar hole was drilled into the corresponding part of the right femur, and a peg, of the same weight, was loosely inserted. While the wound was being closed the bone was accidentally broken across the hole. This, however, was soon repaired, the wound having been completely united.

Three days after, the left femur was accidentally fractured at the seat of the operation. This was subsequently repaired, with an abundant formation of callus.

After a few days the animal regained its health, and remained as well as if nothing had been done.

Three months after the operation the rabbit was killed. In the left leg the peg was found lying imbedded on the surface of a large mass of callus. It was in no way affected. When it was removed, a deep groove, which formed almost a complete canal, appeared on the surface of the callus.

In the right leg the peg was found unaltered, lying loose in a well-formed capsule of connective tissue on the muscles of the thigh under the skin.

There was no trace of abscess, or of any other disturbance of the surrounding parts, in either leg.

Experiment 8.

A hole was drilled through either femur of a healthy young rabbit, and into each a peg, which weighed four grains, was tightly inserted.

The wounds were closed, and soon completely healed, and the animal quickly regained its usual health and condition.

It was killed three months after the operation.

When the left leg was dissected the peg was discovered as it had been placed, transfixing the bone, and standing out about two lines from its front surface. When a gentle attempt was made to withdraw it the projecting portion came away from the rest with very slight perceptible evidence of fracture. There was a ridge of new bone around the orifice.

When a section was made of the femur through the hole, it was evident that a considerable portion of the peg had disappeared, and its now irregular outline could be only imperfectly traced across the shaft, for they seemed to be at some parts continuous. The outline of what remained of the peg could not be defined from the adjacent portion of bone. There was an increased formation of osseous tissue

around, for the margins of the hole were denser in the compact part, and a wall of new bone extended around the remaining portion of the peg across the canal.

In the right leg a very similar state of things was found. The peg was in part absorbed, and the remaining portion appeared upon section to have become here and there blended with the bone. New bone, which was softer than the old bone, was thrown out around the peg, and extended across the canal.

Not only was there no trace of abscess or of any disturbance of the surrounding soft parts in either leg, but the repair was evidently as perfect and complete as possible. The projecting portion of either peg was invested by a capsule of connective tissue.

Experiment 9.

A peg, which weighed four grains, was tightly hammered into a hole drilled into the femur of a young rabbit. The wound was closed, and healed throughout, and the rabbit remained well.

Three months afterwards it was killed.

The peg was found firmly implanted in the bone, and apparently continuous with it. New bone was deposited around the hole, extending from the adjacent surface of the femur upwards on to the peg, where it was gradually lost. There was no trace whatever of any surrounding disturbance.

The bone was not at once divided, as in the last experiment, but was previously macerated.

When a section was made of the femur through the part into which the peg had been driven, very little of the peg could be seen. It had almost entirely disappeared. New bone had been deposited around, so as to form, as it were, a shell, which enclosed all that remained of the peg.

It appears to me that the only explanation which can be offered of the results of these several experiments is that

the absorption of dead bone, when in contact with living bone, is determined by the pressure to which it is subjected.[1]

It is but reasonable to believe that what great pressure may effect in a short time less pressure may accomplish in a longer time.

In some of these experiments, as in the last two, it seems as if absorption of the dead bone goes on only so long as pressure is kept up, while at the same time the irritation produced by its presence is followed by an increased formation of new bone around, which is so closely and accurately adapted to the rough surface of the remaining portion of the dead bone that the line of junction is almost lost.

While, therefore, the possibility of the absorption of portions of sequestra[2] under rare conditions must not be

[1] "The possibility of the absorption of dead bone seems amply proved by cases in which portions of pegs of ivory, driven like nails into bones, to excite inflammation for the repair of ununited fractures, have been removed. The absorption, I say, seems amply proved; but the method of it is made, by the same observations, more difficult than ever to explain; for only those portions of the ivory that were imbedded in the bone were absorbed; the portions that were not in contact with bone, though imbedded in granulations or pus, were unchanged."—Paget. Surgical Pathology, vol. i, p. 472, note.

[2] Hunter says, speaking of the separation of dead bone—"I by no means wish to be understood that no absorption of the dead piece can take place, for, on the contrary, I believe that nature sometimes finds it necessary to the completion of her process; it generally takes place when the separation is slow and the granulating process is quick. This absorption of dead bone takes place in the fangs of the shedding teeth."—Works, vol. i, p. 527.

"Examples of necrosis show that, in the large majority of cases, the separation of dead bone is accomplished entirely by the ulceration or absorption of the living bone around it; but that, in certain cases, especially in those in which pieces of bone, though dead, remain continuous with the living, the dead bone may be in part absorbed, or otherwise removed, not, indeed, in mass, but after being disintegrated or dissolved."—Paget. Surgical Pathology, vol. i, p. 472.

There are cases on record in which dead bone has disappeared without any sensible exfoliation of it. See, for examples, Stanley on Diseases of the Bones, p. 90, &c.

denied—for it cannot be maintained that living parts exercise no influence upon dead bone under any circumstances—the fact that such a result is hardly ever accomplished is easily explained.

It is generally understood that, under certain circumstances, the absorption of living bone may be effected by continuous pressure;[1] for example, we all are familiar with the disappearance of large portions of the bodies of vertebræ by the extension against them of aneurismal and other tumours. But in my experiments there was no satisfactory evidence of the absorption of living bone by the pressure of dead bone.

While, then, the great influence of constant and continued pressure in producing the absorption of living structures is a recognised fact, it may hereafter appear to be at least as important in effecting the absorption of dead ones.

[1] "Bones admit of every mode of absorption that the soft parts do. The most common cause of absorption in them is pressure. Whatever makes an unnatural pressure gives a stimulus for the removal of the bone."—Hunter's Works, vol. i, p. 500.

ON A NEW OPERATION

FOR OBTAINING

UNION OF UNUNITED FRACTURE;

WITH

REMARKS ON ITS APPLICATION TO CERTAIN CASES OF RECENT FRACTURE.

BY

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COMMUNICATED BY

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Received Jan. 16th.—Read March 8th, 1864.

CASE 1.—*Badly united fracture of the inferior maxilla, with extensive deformity of the face, from injury.*

Samuel Smith, æt. 13, was sent to me by Dr. Edwards, of Crewe, in September, 1861. Three months before, the boy had his face entangled in a steam drilling machine, and sustained a severe compound fracture of the lower jaw, with extensive laceration of the left cheek and soft parts surrounding. The lower jaw was broken in two places, between the canine and incisor teeth on the right side, and between the bicuspid and molar teeth on the left. Owing to the injury of the soft parts, it was found impossible to prevent the central detached portion of bone from being

drawn down on the right side, at least half an inch, in which position it united. The following is a report of his state on admission (see Plate III). The lower lip is drawn down at the right angle of the mouth, and the upper one is puckered up at the corresponding part, so that the buccal aperture is of a triangular shape, and cannot be closed by at least an inch and a half on the right side. In addition, there are two very ugly, broad, red cicatrices extending from this part, one downwards to beneath the base of the jaw, and the other across the cheek, nearly as far as its angle. This latter extends through the entire thickness of the cheek, and is firmly adherent to the bone of both upper and lower maxilla, so as effectually to prevent the mouth from being opened in the smallest degree. Three or four fistulous openings exist near the angle of the jaw, and lead to necrosed bone in the position of the fracture on this side. The jaw is immovably fixed. The molar teeth are in contact on both sides, while from the malposition of the central portion the incisors are separated by rather more than half an inch. Firm union of the fracture has taken place on the right side, but on the left, owing to the presence of necrosed bone, the union appears to be imperfect.

To remedy this distressing condition, I proposed, by a plastic operation, to remove the external deformity, and at the same time to replace the bone in a correct position, so that the incisor teeth might be rendered useful for mastication. To accomplish this latter object, I proposed to saw through the bone on the right side at the point of fracture, to raise the central portion of the jaw, and to fix it by some means till union had taken place; but how to fix the bone when replaced was the question. The defective condition of the gums and teeth rendered it impossible that this could be done by fastening the teeth together, either with caps or wires. It was also clear that, owing to the necessary incisions and detachment of the soft parts from the jaw, no external pressure could be borne by structures the ready union of which was of such importance.

It occurred to me that the most ready, if not the best, proceeding would be to peg or nail the two fragments together. I had already observed how little disturbance attended the lodgment of any metallic substance in bone, and I was hopeful that if the fragments could thus be maintained in good position for two or three weeks, I might then withdraw the nails without fear of the deformity recurring.

On the 8th of October the operation was performed. I commenced by cutting out the vertical cicatrix, extending from the angle of the mouth, and then continued the incision beneath the base of the jaw for about an inch, inclining it towards the right side. I then cut out the horizontal cicatrix, and separated the cheek freely from both upper and lower jaw, so as to allow the mouth to open, removing at the same time some small sequestra from the fracture of the left side. Afterwards I separated the integuments from the chin, so as to expose the site of fracture on the right side. The bleeding vessels were secured, and then with a small saw I made a vertical section of the lower jaw through the line of fracture, taking care to divide the bone obliquely from without inwards and backwards. My object in this was to obtain a larger surface for the reunion of the bone, and a broader support to overcome the tendency of the hyoid muscles to draw inwards and backwards the central part of the maxilla. This was effectually and easily accomplished without dislodging the teeth between which the separation of the bone was effected. The sawn surfaces were readily replaced in admirable position, and then with a common sprig-bit I drilled two holes through both pieces of the bone, and, inserting small, smooth, round copper nails, three fourths of an inch long, I drove them nearly home with a hammer. By this means the fragments were readily fixed in good position. The first nail was inserted at a point corresponding with the base of the alveolar process, the second through the dense bone of the base of the jaw. They effectually secured the fragments in perfectly accurate position.

I afterwards readjusted the soft parts of the chin and cheek in such a manner as to remove the deformity of the mouth, and secured the flaps in position by one harelip pin and several wire sutures.

The subsequent history of the case is thus reported:

October 10th.—There has been no constitutional disturbance; eggs, wine, and beef tea have been taken freely; the incisions appear to be free from inflammation, and to have united throughout. Beneath the chin there is rather more hardness and fulness than is desirable, but, with this exception, the progress is as favorable as possible.

11th.—All the sutures were taken out; the incisions having entirely united by the first intention. The bone remained securely in good position.

13th.—Has no pain; the fulness beneath the jaw has disappeared; the patient can open and shut his mouth without disturbing the artificial union of the bone.

22nd.—There is no pain or any sign of inflammation in the neighbourhood of the fracture, which keeps in perfect apposition. The boy can now open his mouth to the extent of three quarters of an inch, and feels the bone so firm that, unless warned to the contrary, he would use his teeth in mastication.

30th.—One of the nails (doubtless the upper one), came away, the boy finding it loose inside the lip.

November 13th.—Discharged cured. The lower nail remains where it was placed; it causes no inconvenience. The soft parts have firmly united over it, and as it cannot be distinctly felt through the integument, and might not be easily discovered on cutting for it, I preferred to leave it alone.

In June of this year (1863) Dr. Edwards informed me the boy continued in good health, and had never had any inconvenience from the presence of the nail, which it was inferred was still in the bone (see Plate IV).

I will now detail a somewhat similar case, in which I adopted the same method of treatment, and the result of

which was so satisfactory that I think we need no longer hesitate to fasten broken bones together in this way when they cannot otherwise be kept in contact. For ages surgeons have recognised the use of the suture in keeping divided surfaces of the skin and soft parts in contact. Why not adopt the same plan with regard to the bones? It will be understood that I make this remark in regard only to those rare and complicated cases in which it is impossible to maintain tolerably accurate apposition of the fractured surfaces, either by the position of the limb or by external support.

Case 2.—A. B—, æt. 28, was admitted under my care on the 30th of March, 1863, having sustained a double fracture of the lower jaw. The bone was broken vertically on each side between the canine and bicuspid teeth, both of which were knocked out on the right side, and partially detached on the left. There was no difficulty, in the first instance, in adjusting the central part of the jaw, and by the application of the ordinary bandage and support there appeared every probability that union would speedily take place; but, after a few days, the man was seized with violent rigors, affecting the whole body, but more especially the muscles connected with the lower jaw. From the effects of these rigors the central fragment became displaced, and notwithstanding the use of various appliances it was found impossible to maintain it in position. The rigors recurred two or three times daily, and at first I was apprehensive were indicative of pyæmia, or blood-poisoning; they were followed by profuse sweating, but the pulse never rose above 80, and there were no other symptoms confirmatory of this formidable complication. Under the influence of large doses of quinine, frequently repeated, the rigors gradually abated, both in frequency and in severity. During the period they continued, which was for upwards of four weeks, it was found essential to remove all mechanical support from the jaw, for every attempt to readjust the fragment was shortly followed by a rigor. The consequence

was that the central portion of the bone became greatly displaced. It was drawn downwards, fully three quarters of an inch, and was turned upon its own axis, nearly across the lateral portions of the jaw.

Before the patient had recovered sufficiently from the exhaustion consequent upon his severe illness to justify me in attempting to replace the fragment, such permanent shortening of the digastric and hyoid muscles had taken place that it was found impossible to lift it into its proper position. Moreover, abscesses had formed on each side in connection with the fracture, and it was obvious that portions of the fractured surfaces had necrosed.

After waiting for some weeks, until the general health had sufficiently improved, I performed, on the 30th of June, the following operation. Enlarging the sinuses leading to the dead bone on either side, I picked out several small sequestra; then, pulling forward the detached central portion of the jaw, I introduced a tenotomy knife immediately behind, and close inside the bone, so as to divide subcutaneously the tendinous attachments of the digastric, genio-hyoid and genio-hyoglossus muscles. This being done, there was no longer any difficulty in replacing the bone in its original position. As, however, from the wounds beneath the jaw, it would have been unwise to rely on any support in this region to keep the fragment in correct position, I adopted another method with a view to this object.

I had in my possession a sprig-bit, which contains within its handle several different-sized drills, any one of which may be fitted to the instrument, and afterwards easily removed. Selecting one about the size of a No. 3 bougie, and holding the central fragment in good position, I pressed it through the skin rather nearer the base of the jaw than the lip, till it came in contact with the bone on the right side, a few lines from the fracture. Then, directing the drill obliquely, so as to transfix the fractured surfaces, I worked it on till its point became firmly lodged in the right lateral fragment; and thus both fragments were securely fixed together in close apposition.. Removing the handle of the sprig-

bit, I left the drill in the bone, with about an inch of its thick end projecting from the chin.

It had been my intention to repeat the same process on the left side, but I found the bone so perfectly secured by the single drill that this was unnecessary.

It was remarkable that immediately after the operation the man could open and shut his mouth with such facility that it was difficult to induce him to preserve a prudent amount of quietness. No pain, disturbance, inflammation, or swelling, followed; the man could articulate and take food better than since he entered the hospital. The wound beneath the jaw rapidly closed, and the bone remained in good position. Scarcely a drop of pus passed by the side of the drill. On the fifteenth day it fell out during sleep, but the bone kept its position; the fractures united, and the man left the hospital, quite well, on the 10th of August.

There are many instances of fractures of the bones in which, I conceive, the same method might be adopted with advantage—cases of oblique compound fracture of the tibia and fibula, that cannot by any contrivance be kept in accurate position—cases in which it has been found necessary to divide the tendo-Achillis, or even to perform the more severe operation of sawing off the broken extremity. In such cases I believe the simple introduction of a drill in the proper direction, so as to fasten the fragments together, would prove efficacious, and, by maintaining perfect rest of the broken ends, greatly facilitate union. The shortening and deformity so frequently resulting would be avoided; and I believe, moreover, the profuse suppuration and inflammation (so often fatal to life), which attends severe compound fracture, would, in all probability, be greatly lessened.

The great difficulty that the surgeon has to contend with in such cases is the spasmodic action of the muscles, which keeps the fracture in constant motion, always irritating the soft parts and increasing their local inflammation. If this

could be overcome, the fatal consequences of compound fractures might often be avoided. A well-directed drill would induce the desired repose far more effectually than any amount of external support, however ingeniously applied. It must not be inferred that I would abandon the use of splints. On the contrary, upon them and upon the proper position of the limb must our chief reliance be placed. I propose only to use the drill as an accessory, and when other means are insufficient.

Let us now to consider the application of the drill as a means of promoting union in more ordinary cases of ununited fracture. From whatever cause union has failed to take place, it is a pathological fact that in a considerable majority of cases there is more or less shortening of the limb, and, consequently, of overlapping of the fractured bones. The fracture may have been simple and transverse, in which case the entire thickness of the shafts overlap to an extent corresponding with the amount of shortening; or it may have been oblique, when, without much shortening, more considerable surfaces of broken bone are opposed to each other. I believe the latter, or oblique fracture, is much more frequently followed by non-union than the transverse, and probably from this reason—that muscular contraction is less opposed, and is thus liable to produce more constant movement of the fractured surfaces.

Whether the fracture has been transverse with considerable shortening, or oblique with less, it is seldom we have much difficulty in making out its direction and the relative position of the fragments. Free manipulation of the limb is not often painful in these cases, and if it is we may overcome this obstacle by the use of chloroform. It is not necessary to make any incision through the soft parts; it is much better to avoid doing so. If, indeed, the line of fracture itself and the ends of the bones cannot be traced by the fingers, its direction may almost always be clearly surmised by attention to the relative amount of mobility when force is applied in different directions.

Let the operator consider carefully these points, and it will not be difficult for him to direct a drill safely through the soft parts to the seat of fracture:—Transfix fairly the superficial fragment, and then drive on the point of the instrument until it becomes firmly lodged in the deeper or more distant fragment. If one drill does not suffice to fix the fragments sufficiently, another may be inserted in a somewhat different direction, so that between the two the bone may be rendered immovable. The handle of the instrument should be removed, and the drills left projecting through the skin. Splints are then put on in the usual way, and the limb kept in a proper position. No local application is required.

After three or four weeks the drills may be removed; the punctured wounds heal readily, and experience proves, as far as this plan has yet been tried, that firm union takes place.

In performing this operation some caution is desirable not to allow the drill to penetrate beyond the limits of the deeper fragment for, by doing this, not only would the bone be less secured, but there would be risk of injuring important structures beneath, such as blood-vessels and nerves; and, again, I do not think it would be prudent to direct the drills, supposing two are inserted, so that they should at any part of their course approach within three quarters of an inch of each other. Such close apposition I should fear might give rise to more inflammatory action than is desirable, and might possibly determine suppuration or necrosis.

But supposing the relative position of the fragments is not such as I have described—supposing there is no overlapping, either from obliquity of the fracture or from the entire thickness of the shafts resting against each other—that, in short, the fracture has been transverse and the extremities are in good position—yet no union has taken place. How, then, it may be asked, can it be possible to fasten the fragments together? I have not yet had an opportunity of putting in practice the operation I would advocate in such a case; I believe such instances rarely occur, yet in

the femur and the humerus we sometimes meet with it. Even then, I think, the drill might generally, with a little care, be directed so obliquely as to traverse and penetrate both fragments.

It may be urged, and, doubtless, correctly so, that this could not always be accomplished, for the broken ends of bone resting in apposition, and yet ununited, become rounded off and more or less conical from absorption, and sometimes, moreover, a synovial sac is formed in the substance of the uniting fibrous tissue. Probably the simple insertion of the drill into each extremity of the bone, after the manner of Dieffenbach, but without any external incision or exposure of the bone, would set up sufficient irritation to produce union. If it did not, and failure resulted, I think it would be legitimate and a safer operation than resection to break down by extension and manipulation the connecting fibrous union, so as to make the fragments overlap, and then, after swelling had subsided from absorption of the effused blood, to fix the bones with drills as previously described. Of course, considerable shortening would be the consequence, but so it would have been had the ends of the fragments been cut out.

Having made these observations, I will now add, in support of the views I have advanced, the following cases.

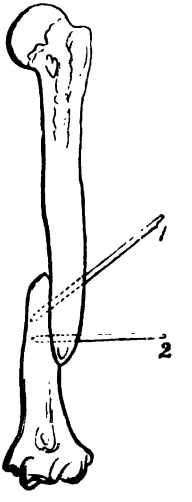
They have all been under my own observation, and are kindly given to me by my friend, Mr. Fletcher, in whose practice they occurred.

Case 3.—S. M—, æt. 44, a strong and healthy labourer, was admitted into the Liverpool Workhouse Hospital on September 23rd, 1861. About a year previously he had broken his left humerus. The limb had been placed in splints, and treated in the ordinary manner, and he had, I believe, considered himself cured. On admission he was found to have an ununited fracture at the junction of the lower with the middle third of the humerus. The lower fragment was drawn up, so that it was overlapped by the upper one and lay on its inner side. The outline of the bones could be

distinctly felt; there was no thickening of either, and no attempt at union. The arm was placed in splints, and kept so for about twelve weeks, but without good result.

On December 27th I performed the following operation, in which I was kindly assisted by Mr. Bickersteth. I passed a tenotomy knife through the integuments, &c., down to the bone, on the outside of the arm, as nearly as I could judge, an inch above the end of the upper fragment. The bone here appeared softened, as the knife passed easily through a considerable portion of the upper fragment (see fig. 1). Next, I passed a drill-head as far as the wound made by the knife would allow, and, as soon as its point was fairly against the solid bone, I worked it on by means of a common Archimedean drill until I was sure that it had traversed the whole thickness of the upper fragment, the outer surface and the medullary cavity of the lower fragment, and had fixed its point in the hard bone of the inner surface of this fragment. In consequence of the softness of a portion of the bone, this did not hold the two pieces firmly together. I therefore passed a second drill-head obliquely through both of them, piercing the integuments at a point about an inch and a half above the former one. This held them well together. The arm was carefully placed in splints, with the elbow bent at a right angle. The patient was kept in bed, the arm being slightly raised on a pillow. He experienced but little inconvenience from the presence of the drills. On January 2nd there was some suppuration around them, and the lower one was removed. The upper was allowed to remain for a week longer. After this there was some inflammation, a thickening around the punctures, and at one time matter appeared

Fig. 1.



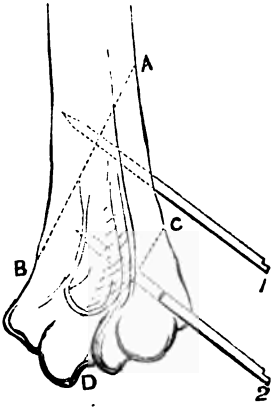
to be forming under the fascia; all this, however, subsided without requiring any other vent than that afforded by the punctures through which the drills had been passed. The arm was kept completely at rest till the commencement of April, when he was allowed to move the elbow-joint, which he was soon able to do with ease. Some troublesome stiffness of the shoulder, resulting from the constrained position in which the arm had been kept, remained up to the time of his discharge. He left the hospital on May 21st, 1862, completely cured.

Case 4.—M. C—, æt. 44. Admitted May 15th, 1862. Received a compound comminuted fracture of the left humerus and simple fracture of both bones of the forearm in August, 1861. On admission there was still a small portion of the wound over the internal condyle suppurating; from this a small piece of bone was removed, and it soon healed.

The fracture of the forearm was well united, but there were two fractures of the humerus still quite loose, one of them passed obliquely forwards and inwards from about four and a half inches above the external condyle to just above the internal condyle, thus separating it from the shaft of the bone; the line of the other fracture passed from about two and a half inches above the external condyle obliquely into the joint, thus detaching the external condyle. Each condyle was thus movable independently of the other, and both independently of the shaft of the humerus. When an attempt was made to bend the elbow the only mobility which was found was between the upper fragment and the shaft of the bone. On June 9th the drills were passed in the same manner as in the preceding case (see fig. 2). The first was directed from the middle of the shaft of the bone from the outer side, inwards and upwards, thus pinning the upper fragment to the shaft. The second was passed from the front of the lower fragment upwards, inwards, and backwards, thus uniting this piece of bone to the one above it. Still less pain and irritation were occasioned in this case by the presence of the drills than in the

former one. The upper drill was removed on June 24th; it was then loose, and there was slight suppuration around it. The second was removed on July 1st; this was somewhat tightly held. On July 23rd I examined the elbow carefully, and found that the joint could be moved easily, and that the fragments of the humerus were firmly united to each other and to the shaft of the bone. He left the hospital on August, 18th at his own request. I was anxious to have kept him for three months under observation, but he wished to return to his work. I have not seen nor heard of him since, from which I infer that he has remained well.

Fig. 2.

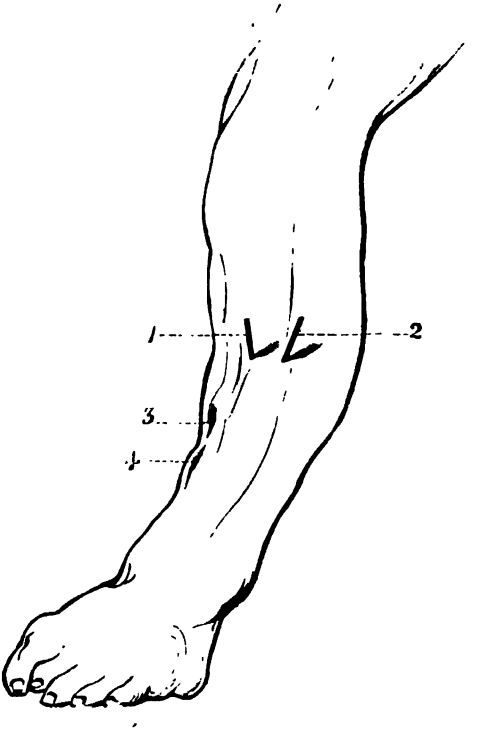


Case 5.—J. M—, æt. 26. Fractured the left tibia and fibula at Aden, in October, 1859. He had fever after the accident, and recovered with the leg very much curved. He walked very well until he was attacked by typhus, for which he was admitted into the Liverpool Workhouse Fever Hospital on May 22nd, 1863. He passed favorably through the fever, and was transferred to the surgical wards on June 22nd. The tibia was found to be in two portions, separation having occurred at the old fracture. The fibula appeared to be entire. There had been suppuration, which appeared to have commenced at the site of the fracture, and there were two small sinuses opening on the inner surface of the leg. The limb was placed in gutta-percha splints. The sinuses soon healed, but union did not take place between the fragments of bone.

On July 14th two drills were passed through the adjacent ends of the fragments of the tibia, the inner

one being directed downwards, backwards, and outwards (see fig. 3); the other downwards and backwards. A wooden splint was placed upon the outer side of the leg.

Fig. 3.



15th.—Suffers very little pain.

18th.—Some swelling of the parts around the drills.

19th.—Rather more swelling, and ichorous discharge from the situation of the old sinus.

20th.—A small abscess was opened and the bandages below the wound were loosened.

22nd.—Swelling much less; drills still firm; another small abscess was opened.

30th.—The outer drill (No. 2) was removed; and some little force was required for its extraction.

31st.—The inner drill (No. 1) was removed; this was quite loose.

Gutta percha splints and bandages were applied until September, when union was found to be complete.

When seen on January 7th, 1864, the union of the bones was perfect. There was a slight superficial ulcer still remaining about the site of the collections of pus previously mentioned. He could not place his foot so flat upon the floor as before the operation; but this was chiefly due to some stiffness of the ankle, arising from the joint having been kept motionless for so long a period of time.

DESCRIPTION OF PLATES III AND IV.

PLATE III.—Case of Samuel Smith, before operation.
„ IV.— Ditto after operation.

CASE OF SAMUEL SMITH.

BEFORE OPERATION.

[illegible] from Photo.

CASE OF SAMUEL SMITH.

AFTER OPERATION

Engd by Bocklett & Edmt from a Photograph

W. West & Son

ON A NEW METHOD

OF PROCURING THE

CONSOLIDATION OF FIBRIN IN CERTAIN INCURABLE ANEURISMS.

BY

CHARLES H. MOORE, F.R.C.S.,

SURGEON TO THE MIDDLESEX HOSPITAL.

WITH THE

REPORT OF A CASE IN WHICH AN ANEURISM OF THE ASCENDING AORTA WAS TREATED BY THE INSERTION OF WIRE.

BY

CHARLES MURCHISON, M.D., F.R.C.P.,

PHYSICIAN TO THE LONDON FEVER HOSPITAL.

Received Feb. 4th.—Read March 22nd, 1864.

In the following joint paper the theory of the proposed operation is first described by Mr. Moore. Dr. Murchison furnishes the report of the entire case, with the medical comments upon it. For the concluding surgical remarks Mr. Moore is alone responsible.

I.

As the proposition of a new principle in surgery, and the extension of practice by means of it into a new region of disease, must necessarily undergo discussion, it would have

been advantageous that the principle to be enunciated in this paper should have been further tested before being made public. But the notoriety which the proceeding has obtained, and the inexact reports which have been circulated respecting it, induce me to bring the facts forward earlier than I should otherwise have done. Before the tribunal of the Royal Medical and Chirurgical Society, however, I have every confidence that the principle will be judged apart from the incidents of failure attending its first application. It will be convenient to throw my first remarks into a narrative form.

On the 20th of February, 1863, my colleague, Dr. Henry Thompson, requested my attendance on a case of aneurism of the thoracic aorta which was under his care in the Middlesex Hospital. Notwithstanding all medical treatment, the aneurism was increasing in size, and it had recently protruded on the right of the sternum, in the second intercostal space. In sending for me, Dr. Thompson desired to have the opinion of a Surgeon on the propriety of employing galvano-puncture in the treatment of the case. I stated that I feared that operation would shorten instead of prolonging the patient's life.

As this decision implied the abandonment without help of a disease which, if situated in an extremity, would be accessible to various means of cure, I reviewed carefully the circumstances of a thoracic aneurism, in the hope that there might be some special condition which, in the present state of our knowledge, would suggest a method of relief. To cure—that is, to close artificially—the cavity of an aortic aneurism might well be deemed impossible; yet the following considerations appeared to afford a reasonable ground for hope that benefit might even in such a case be conferred by surgical means.

It was plain, at the first thought, respecting the circumstances of an aortic aneurism, that no treatment could be applied to the artery. Our remedial measures are, consequently, limited to such as may effect changes within the aneurism itself.

For this purpose no treatment is of any value which does not provide for the separation and adhesion of fibrin. It may suffice for the cure of some aneurisms of the extremities to produce consolidation of the entire mass of the blood contained in them, but in those of the aorta any substance except adherent fibrin would certainly be broken up or displaced by the force of the currents. The *first* indispensable condition, therefore, for the cure of a thoracic aneurism is to provide means of eliciting fibrin from the blood.

Perhaps there are no aneurisms in which the deposition of this material is naturally more abundant than in those of the aorta. They are sometimes lined by strata of fibrin of very great thickness. But as the accumulation of it is not always thus abundant, and is too often partial, the wall of the aneurism is either not sufficiently or not equally and uniformly supported, and it yields at some weak spot to the violent impulse with which the blood bursts in from the close proximity of the heart. The power of collecting solid fibrin possessed by such aneurisms is inadequate to effect a cure of them. Some means must be supplied to them for increasing the quantity of adherent lymph where it is insufficient, and for procuring its deposition where it is wanting.

Now, ample as is the supply of fibrin in the blood, and its disposition to separate from the other constituents of that fluid, there is nothing in an aneurism on which it can settle except the wall; but any quantity which may collect on it is almost invariably insufficient to resist the expansive force of the arterial current. In the centre of an aneurism there is, meanwhile, a space full of blood, with fibrin ready to consolidate upon any apt material. A *second* indispensable condition for relieving an aortic aneurism is, therefore, to extend the surface within it on which fibrin may coagulate.

On combining these two requisitions for the solidification of an aneurism which cannot be treated through the artery, I came to the conclusion that both might be met by one proceeding, namely, by inserting some foreign body into the cavity of the aneurism. The second condition, that of

exposing a broader surface to the blood, would, if attained, ensure the attainment of the first, or the separation and consolidation of fibrin; and from what is known of the manner in which that substance coagulates, it is clear that the rapidity of its deposition would be proportionate to the roughness of the new surface provided for its attachment.

I was familiar with the fact that fibrin will accumulate in large quantity upon a foreign body placed in a current of arterial blood, having already published some observations on the subject in Holmes's 'System of Surgery.' As they are pertinent, and, indeed, of much importance to the theory I am propounding, I venture to quote them from the essays on "Injuries of Arteries," and on "Atheroma," in the first and third volumes of that work.

"Mr. Laugier presented to the Anatomical Society of Paris, in 1849, the following remarkable case:—A healthy young man died with a needle in the left ventricle of his heart, and with gangrene of the left leg. The needle was found fixed in the apex of the ventricle, with its point directed towards the aortic orifice, and covered with 'decolorized, sanguineous concretions, very coherent and irregular,' which were prolonged into the aorta. The lower end of the aorta and the iliac arteries, on both sides, were filled with an adherent clot. It seemed plain that the plugging of the arteries, and the consequent gangrene, had their origin in the detachment of fragments of the lymph, which had first adhered to the needle."—Holmes's 'System of Surgery,' vol. iii, p. 333.

"No. 1565*a*, in the Hunterian Museum, is a specimen of the ascending aorta of a man, laid open, and disclosing, in its interior, a large jagged mass of skin and subcutaneous tissue, and a hemispherical socket of lymph, adherent like the skin to the interior of the aorta. Out of this socket a spherical bullet, also covered with lymph, has fallen to the bottom of the vessel. The piece of skin is in contact with the lining membrane of the ascending aorta, about an inch above the valves. The socket of lymph is fixed to the

artery immediately beyond the skin. The rest of the artery is healthy. The account of the case given by Mr. Brunton, Assistant-Surgeon on board the hospital ship in the Mediterranean, and communicated by Sir Stephen L. Hammick, is to the effect that a boat's crew, detached to cut out a vessel, met with determined resistance. Amongst the wounded was a seaman, who affirmed that a musket-ball, striking his oar, had run along it and entered his side. He lost a good deal of blood at the time, and then, almost completing the third day from the injury, died."—Holmes's 'System of Surgery,' vol. i, p. 669.

These facts occurred to my recollection in the course of the foregoing reflections, and they left no doubt on my mind that, if a suitable material for introduction into an aneurism could be found, the deposition of a large quantity of fibrin upon it might be ensured.

The foreign body which our present experience shows to be least irritating to the natural tissues is wire. Its innocuousness when employed for sutures is daily observed, and particularly in contrast with silk, which readily induces suppuration. Wire, moreover, appears adapted to the object in view, since it would present an extensive surface for the accumulation of fibrin in the central part of the aneurism, would be likely to attract the fibrin, as twigs do in the experiment of whipping freshly drawn blood, and would detain and support the fibrin when attracted. If a large quantity of wire could be introduced into the interior of an aneurism, and disposed about it in coils, a corresponding quantity of fibrin would soon accumulate and increase upon it. Progressively, a cure might result by the union of the central and mural lymph. The wire would remain in the aneurism, enveloped in clot, and be harmless.

For such treatment to be of service, the aneurism must be accessible from the surface of the body. It could hardly be employed in an aneurism of the abdominal aorta or of the thoracic beyond the arch.

Moreover, no artery should open from the aneurism; it should have but one aperture. Were the wire exposed in a

violent current of blood, fibrinous clots would certainly break off from it and plug distant arteries. An aneurism at the root of the neck would therefore require the previous performance of Brasdor's operation; one of the aorta should be a sacculated, not a fusiform, one.

In this, indeed, in any case, the chief danger would consist, namely, that the fibrin, being at first fragmentary and loosely attached to the wire, might break off, or might regurgitate through the aperture in the artery and then break off. This danger must obviously be proportioned to the size of the spaces between the coils, and it would diminish with the quantity of wire used. The chief fault, if failure occurred from the manipulation, would doubtless be not introducing enough wire.

Another danger might arise from the projection of a coil or end of wire into the artery through the aneurismal opening, which would lead to the breakage of fibrinous strings and their propulsion along the stream. It would hence be important to ascertain the position of the aperture of communication with the artery, and to avoid it in introducing the wire, as well as to use wire which would not bend within the area of an artery.

Upon considering what might be expected to become of the wire, it appeared to me—1. That if completely coated with fibrin, it would be a pretty firm skeleton-like support for the soft lymph. In course of time it might with the lymph be hammered out against the wall of the aneurism, when it would form a firm sieve-like layer, lining the sac and materially strengthening the wall. 2. If iron were selected, and it should become partly uncovered, it might eventually be dissolved. 3. It might move with the blood, until a flexure of it intruded into the artery. This was likely to be avoided by using fine wire, which would be little stirred by either current, to or fro; while, if it all became coated, the force of both currents would be lessened directly as the coating thickened. If bulky wire were used, though of silver, it would be difficult to tangle, and would resist the subsequent collapse of the sac after the consolidation of its

contents. The delicacy of the wire should bear some relation to that of the lymph it is to ensnare and to support.

The method of performing the operation.—Choose wire which is stiff enough to pass the canula without bending, but so fine as to bend easily when pressed against the wall within the aneurism. Provide a straight, short canula of thin silver, sharply pointed at one end, expanded at the other, and let its tube be somewhat larger than is sufficient to give ready passage to the wire. Let there be also a thicker wire, which fills the canula, and is flattened at the end. This is for use as a probe.

Puncture with the canula some part of the aneurism which shall not be opposite to the presumed situation of the aperture from the artery. Introduce the probe, and ascertain the position and size of the arterial opening, as well as the dimensions of the aneurism itself. This would determine the directions in which the fine wires should be introduced, and the quantity which the aneurism might be expected to hold. Push in the fine wire, directing its point against the opposite wall of the sac, and, whilst introducing more wire, divert the end of the canula. The wire must bend from its fixed extremity in a curve determined by the direction given to the canula. Continue to introduce wire and to move the canula, and coils of wire will be formed of any required shape, and in any part of the sac.

In withdrawing the canula the wire must not be suffered to remain in the puncture. If it did so, the issue would probably be fatal hæmorrhage. The last part of the wire must be pushed fairly into the aneurismal cavity with the flat end of the probe. The canula may then be withdrawn upon the probe, on the removal of which, last of all, the skin would slide back again over the tumour, and the puncture in the sac would not correspond with that of the skin. The puncture might be covered with collodion.

I put my proposal in writing the same evening, and the next day communicated it to my colleagues, Mr. De Morgan

and Dr. Henry Thompson. I had no opportunity, however, of putting it in practice in the case by which it was suggested, as the patient, on the 21st of February, quitted the hospital without leave.

No new example of the disease in a hopeless state presented itself to me until November, 1863, when I was requested by my colleague, Dr. Murchison, to see an advanced case of thoracic aneurism which was under his care in the Middlesex Hospital.

The proposed method of treatment being untried and incapable of correction and proof by experiments on the lower animals, I did not press for its adoption so long as the patient's life was likely to be prolonged without it. In January, 1864, however, changes in his state had occurred which appeared to justify the operation. These will be best understood by Dr. Murchison's report of the case.

II.—*Report of a Case of Saccular Aneurism of the Ascending Aorta projecting through the Anterior Wall of the Left Side of the Chest.* By CHARLES MURCHISON, M.D.

Daniel D—, æt. 27, became a patient at the Middlesex Hospital, under my care, on November 10th, 1863. Eight years before, he began to suffer from palpitations and dyspnœa, and after some months he had an attack of hæmoptysis. The hæmoptysis recurred at intervals, and in November, 1862, he first noticed a pulsating swelling in front of the chest, to the left of the sternum. This increased with considerable rapidity, and the patient now became subject to severe attacks of angina pectoris. At the time he first came under observation, the tumour was situated in the angle formed by the left clavicle and the left margin of the sternum; it measured ten inches in circumference at its base, and projected about two inches from the wall of the chest; its surface was rounded, and tolerably uniform, except at the upper part, where there was a tendency to point. Over the whole surface of the tumour dis-

tinct pulsation could be felt, each beat corresponding to the impulse of the heart. Nothing resembling an aneurismal bellows-murmur could be heard; but both cardiac sounds, and particularly the second, were louder over the tumour than at the base of the heart. There was dulness on percussion to the right of the tumour, over a space measuring two inches transversely and three inches from above downwards. The apex of the heart could be felt beating between the fifth and sixth ribs. The cardiac dulness was slightly increased, but the sounds heard on auscultation were normal. There was no inequality of the arterial pulse on the two sides of the body. The voice was normal. The patient had an occasional cough, and expectorated a viscid mucopurulent matter, but there was nothing peculiar in the character of the cough. Over the whole of the left side of the chest there was comparative dulness on percussion, with harsh, at some places tubular, breathing, and subcrepitant râle. On the right side of the chest the percussion was clear and the breathing puerile. The appetite and digestion were good, and the bowels regular. There was no dysphagia, and no pain or tenderness at any part of the spine. The pupils were of normal and equal size. On November 20th, and again on December 28th, the urine was ascertained to be free from albumen.

After the patient was admitted into the hospital, on the 20th of November, the attacks of angina almost entirely ceased. The pulse varied from 104 to 116 when the patient sat up, but would fall to below 100 when he lay down. The size of the tumour continued to increase, until, on the morning of January 7th, 1864, it measured sixteen inches and three quarters at its base, and projected two inches and two thirds from the wall of the chest. The tendency to point at its upper part became more decided, and the integuments at this part were much attenuated, and assumed a dusky red discoloration, while occasionally they were the seat of pricking pains, and were slightly tender. These changes were most marked during the last week of December and the first week of January. The patient's

general health, notwithstanding, did not suffer. He ate and drank well; he got up daily, and walked about the ward. On the evening of January 6th he played a game of draughts with another patient, and on the following morning he was up and walking about as usual.

Early in January it became obvious that the bursting of the aneurism through the integuments could not be long delayed. It was accordingly resolved to recommend to the patient Mr. Moore's proposed operation. During the month of December this operation had been carefully considered, but it was then deemed unadvisable to have recourse to it. It was now explained to the patient that the procedure in question offered some chance of prolonging his life, although in itself it was not free from danger. The patient at once assented, and the operation was performed on January 7th, at half-past 1 p.m.

The operation consisted in the introduction of a quantity of fine iron wire into the aneurism, with the object of inducing coagulation. A small pointed canula was inserted into the tumour, and the wire was passed in through this without difficulty. The operation occupied one hour, and the quantity of wire introduced was twenty-six yards. It gave rise to no pain or inconvenience excepting a slight and transient feeling of faintness. The quantity of blood lost did not exceed half a fluid ounce.

The immediate effects of the operation were a reduction of the pulse from 116 to 92, an almost complete cessation of the pulsation in the tumour, and a diminution in its size. Immediately before the operation the circumference of its base was sixteen inches and three quarters; at the close of the operation it was sixteen inches. These changes began to be noticed soon after the commencement of the operation, and became more marked as it was proceeded with. At a quarter past 11 p.m. the patient was asleep, and his pulse was only 78. He slept comfortably during the night, and had no bad symptom until the following morning.

On January 8th, at 9 a.m., the patient was seized with

a fit of rigors lasting three quarters of an hour, and followed by great pain in the back of the neck, and some pain in the tumour. At 1 p.m. the pulse had risen to 144, and was full and bounding. The action of the heart was tumultuous, and all the arteries of the body could be felt throbbing with considerable force, but there was no difference in the force or volume of the beat on the two sides of the body. The patient complained of great pain in the tumour when he moved. The tumour was already somewhat larger than before the operation, and the dusky discoloration was of a deeper tint. There was intense thirst and great restlessness, the skin was dry and very hot, and the respirations were 40. At twenty minutes past 1 p.m. the patient was bled to the extent of eighteen ounces, and at half-past 3 p.m. twelve ounces more blood were abstracted. After the second bleeding twenty minims of Battley's sedative solution were administered.

From these measures the patient derived temporary relief, but he had a restless night, and at 6.30 a.m. the following morning (January 9th) he had a second attack of rigors. At 1 p.m. he had a third attack. At 1.30 p.m. he was in great distress, owing to pain referred to the tumour and to the back of the neck. The tumour was extremely tense, and decidedly tender, particularly at its upper part. Its circumference at the base measured an inch and three quarters more than before the operation. Distinct pulsation could be felt again at its upper part. The pulse was 136, and soft; the action of the heart was less tumultuous, and there was no abnormal cardiac sound. Large and repeated doses of opium and digitalis were now commenced. At 10.30 p.m. the pulse had fallen to 126, but the patient complained of being afraid to cough, on account of a severe jerking pain in the tumour, which the effort to cough always induced.

On January 10th, at 10 a.m., the patient had taken seventy three minims of tincture of digitalis, and the equivalent of almost ten grains of opium, during the preceding twenty-one hours, and the result was that he was in

less pain, the pulse had fallen to 104, and the tumour was slightly reduced in size, its circumference being half an inch less than on the preceding day. At 7.30 p.m., however, he was seized with a severe burning pain in the tumour, and a feeling of tightness, as if it were going to burst. The tumour was larger and more tense than ever; the pulse rose to 132; the heart's action was again more impulsive, and there was intense thirst. To-day it was noted for the first time that the pulses in the right temporal and radial arteries were slightly fuller than in the corresponding vessels on the left side.

Opium in large and repeated doses, along with digitalis, was persisted with. In the course of two days and a half (commencing on January 9th) as much as the equivalent of twenty-seven grains of opium was administered. The treatment, however, failed to give relief. The tumour increased rapidly in size, and on the 11th distinct pulsation could be felt at several parts of its surface. The radial pulse was 128, small and compressible, and still fuller on the right side. The beat of the right anterior tibial artery was also decidedly fuller and stronger than that of the left. The cardiac impulse was extremely feeble. The respirations were performed chiefly by the diaphragm and the muscles on the right side of the chest; the left side of the chest was almost motionless. The whole of the left side of the chest in front, unoccupied by the tumour, was dull on percussion, and no respiratory sound could be heard on this side, except immediately below the clavicle. Brandy and other stimulants were now given, but without any decided result.

On the morning of the 12th the patient was evidently sinking. The pulse was about 136, but was so weak as to be counted with difficulty. The circumference of the tumour at its base was now three inches and a half more than before the operation, and the urine passed during the night was found to be loaded with albumen. At 11 a.m., four days and twenty hours and a half after the operation, the man died.

An autopsy was performed a few hours after death. The walls of the external tumour were formed by the integuments and fibres of the pectoral muscle, infiltrated with serum. They were nowhere less than a quarter of an inch in thickness. The skin covering a great part of the tumour presented a deep livid hue. The interior of the tumour was filled, for the most part, with a fibrinous coagulum, enveloping and imbedded in the coils of wire, and firmly adherent to the surrounding walls. The rest of the cavity contained fluid black blood. The interior of the outer tumour was nowhere lined with a prolongation of the arterial coats, but it communicated with the proper aneurismal sac within the chest by two large openings in the first and second left intercostal spaces, the intervening rib being bare and eroded, and at one place broken through. The aneurismal tumour within the chest was about the size of a man's fist. It lay immediately behind the sternum; it encroached slightly upon the upper lobe of the left lung, and inferiorly it rested upon the right auricle. It was partially filled with a fibrinous coagulum, which was continuous with that in the outer tumour, and was adherent at one part over a space measuring about one third of an inch in diameter. None of the coils of wire had entered the inner sac. The inner surface of the sac was rough from the presence of firmly adherent patches of fibrinous deposit. The sac communicated by a circular opening, scarcely so large as a sixpence, with the ascending aorta. Through this opening a clot projected from the aneurism into the vessel, and extended upwards into the arch. The greater part of this clot was evidently of a post-mortem date; but part of it, close to the opening, was pale, firm, and laminated. There was considerable atheroma of the coats of the thoracic aorta. The pericardium contained about eight ounces of turbid serum, and its opposed surfaces were coated with a thin layer of recent lymph. The upper part of the parietal pericardium presented a patch of livid discoloration, about the size of a florin; and at the centre of this patch the cavity of the pericardium was merely separated

from that of the aneurism by a delicate membrane. It was at this part of the aneurism that the coagulum was adherent. The heart was slightly hypertrophied. Its valves and muscular tissue, and likewise the coronary arteries, were healthy. The left lung was everywhere firmly adherent, and its pleura much thickened. On section, numerous cavities were observed, evidently resulting from dilatations of the bronchial tubes. In the intervening spaces a firm fibrous tissue took the place of the normal vesicular structure. The right lung was for the most part healthy. Both kidneys contained a number of circumscribed abscesses, varying in size up to that of a small pea, and containing characteristic pus corpuscles. A cluster of six of these small abscesses was found at the apex of the left kidney. Each abscess was surrounded by a zone of vascular injection. In the cortical substance of both kidneys a number of patches of yellowish deposit, of a larger size, but less defined outline, were also observed. On microscopical examination, this appearance appeared to be due to the presence of a granular exudation deposited between the uriniferous tubes. The liver was large and fatty. The other parts of the body could not be examined.

III.—*Surgical Remarks.* By C. H. Moore.

I purpose limiting my remarks on this case to the operation, with its results, and to a review of the theory on which it was founded.

The operation was attended with no difficulty, and, excepting the prick of the canula, with no pain. The contact of the wire with the interior of the sac at first occasioned neither soreness nor inconvenience, no sensation even being produced by it but that of the creaking of the coils against one another. The wire passed in easily, meeting no obstacle to its introduction until many coils had accumulated, and, large as was the quantity inserted, none of it was pushed through the opening in the wall of the chest.

This last fact entirely removes the fear of one of the dangers for which I had prepared, namely, the entrance of a coil into the canal of the artery. No opening from an artery into an aneurism is likely to be so wide as that which connected these two sacs. The other principal danger which had appeared likely to be incurred was, perhaps, not entirely escaped, since some minute fragments of the fibrin may have been detached from the main clot, and propelled along the current of the arteries. To such impacted fragments the recent changes in the kidneys may have been due, but there was no other distinct sign of their existence in any part of the body, and in the incomplete examination of it which was made none were detected with the naked eye. Further, the minute puncture in the aneurism healed with little difficulty, although the canula had remained and had been moved freely in it for the period of an hour. The hæmorrhage was slight.

The immediate result of the operation, and the early progress of the case after it, afforded a confirmation of the theory which was even more satisfactory than had been looked for. The external aneurism, when first probed, gave the impression of containing a small quantity of very loose clot near its surface, and this clot probably prevented the issue of a jet of arterial blood through the canula, but it was so loose as not perceptibly to obstruct the entrance of the flexible wire, and it certainly in no degree interfered with the pulsation of the aneurism. At the end of an hour, however, notable changes had taken place; the aneurism had ceased to beat, it had lessened in size, and the pulse, which had ranged at about 112 for weeks, and which reached 120 before the operation, subsided to 92 and at night to 78. It could hardly be doubted that a large mass of fibrin had been already entangled amongst the coils of wire, whilst the rapidity and completeness with which the action of the heart was subdued, appeared in striking contrast with the previous failure of medicines to reduce the pulse. It showed, moreover, that the effect of the introduction of the wire was in the first instance not inju-

rious, but tended towards health and cure. It is important to dwell on these facts, because their value, though in no degree altered, may be obscured by the course which the case eventually took.

The influence of the operation upon the life of the patient was twofold. During the few days, and especially for twenty-four hours, preceding the operation, very rapid changes were occurring in the external tumour. The changes were such as indicated the approaching rupture of the aneurism, and these the operation arrested; but while it averted imminent death in one way, in another it appeared to provoke a painful inflammation, which proved fatal by involving vital parts.

The manner in which the operation actually determined the fatal result is far from being clear. In circumstances so unprecedented it is difficult to discern what was the actual sequence of the morbid events from the morning after the operation until the patient's death; but the early outbreak of the symptoms, and their distinct connection with the chest, associate their origin with the external tumour and its new contents.

Whether the first inflammation was induced by the presence of the wire, or, independently of the wire, by the accumulation through mechanical means of so large a mass of fibrin, is a question of no less interest than obscurity. Before the occurrence of this case we possessed no facts demonstrating the effects of a rapid and copious deposition of fibrin from the blood without previous inflammation. It now appears probable that inflammation will arise as an early local consequence of it, and a constitutional effect be also produced which is manifested in acute febrile symptoms of proportionate severity. While closely watching the progress of an aneurism, I have observed it become red, hot, and tender, without sufficient variation of the circulation of the limb to account for the change, and have conjectured that the symptoms arose from the recent addition of a layer of fibrin on the wall. If this explanation should prove to be correct, there was enough in the unlooked-for abundance of the fibrinous mass in this

aneurism to account for the local inflammation, as well as for the constitutional excitement.

Two principal pathological changes followed this first inflammation, viz., acute inflammation of the pericardium, and of portions of the kidneys. Neither of these changes appears to be clearly traceable to its cause. The pericardium was not continuous with the inflamed external tumour, and the inner aneurism, with which it was in the closest contact, which had indeed almost burst into it, was uninflamed. Both pleuræ also were free from inflammation. The coronary arteries contained no fibrinous impacted plugs.

The state of the kidneys may be ascribed to the introduction of fragments of fibrin into them from the aneurism with the arterial current, and I am not prepared to deny that this may have taken place. The certainty of the occurrence, to any large extent, would render the operation improper. But it appears to me premature to conclude either that it did occur to a fatal extent, or that it positively did happen at all. It is to be observed that similar isolated inflammations had taken place in these kidneys many times before without a fatal result; that such appearances are not usual in kidneys to which detached fragments have been distributed from diseased mitral valves; and that they are equally intelligible as a result of the activity required of renal organs, already so scarred and obstructed, upon the occurrence of a severe inflammation in a distant part.

However the extra-thoracic inflammation may have originated, whether in the tissues about the fibrin or with the fibrin itself, both it and the general symptoms must equally be ascribed to the operation. Not so, however, the pericarditis. That appears to have been an indirect result of the operation, a mechanical consequence of a febrile excitement of the circulation. It was, as such, not an inevitable or an essential result of the operation, and it proved fatal only because of the incidental connection of that aneurism with the pericardium. The theory, nevertheless, was still proved

to be erroneous, in so far as it included the presumption that a foreign body of the quality and size which was inserted into this aneurism would be harmless. By it or by the clots containing it inflammation was set up. But that inflammation was limited to the immediate vicinity of the foreign body; it was in itself not mortal, and it afforded no proof that in an uncomplicated case a patient might not recover with benefit from the operation.

It remains to inquire what encouragement to repeat the operation is furnished by the case which has been related, and with what modifications, if any, it may be practised in future.

If our judgment on these questions must be decided by the fatal issue of this single case (notwithstanding or because of the performance of the operation), or by the painfulness of the mode of death—and still more if in the original idea of the operation we can fix upon some condition of inevitable failure—the attempt is needless to improve the method which I adopted in the endeavour to save this man's life. But if there be no manifest and essential fault in the proceeding, I cannot think it right to abandon an operation, which was so promptly followed by the most marked and positive improvement. On the contrary, I regard its early results as strikingly confirming the main part of the theory, and as affording for other cases a substantial promise of advantage, from which future thought has but to eliminate the cause of ill success. The very condition which it is our chief desire in the management of an aneurism to obtain was here obtained, since within an hour a large quantity of fibrin had accumulated.

Two other circumstances in the present case confirm my hope of eventual success. Of these one is the firm adhesion which was found to have taken place between the fibrin enclosing the wire and the wall of the aneurism. The adhesion was inseparable without dissection, and the mass of wire and fibrin together was only removed by tearing through the latter. Some future observation may disclose the period at which this adhesion may have taken place. If it

should be found to occur soon after the introduction of the foreign body, the fact will enable us to dispense with the permanent support of the wire. The present case, however, leaves that question undetermined, as the clot was found thus adherent in an inflamed sac, and on the fifth day from the operation.

The last valuable fact is the exemption of the intra-thoracic part of the aneurism from inflammation; or, if it were really inflamed, the far slighter severity of the process in it than that exhibited by the outer sac. The latter was lined by no smooth membrane, and the rough tissues around it were found full of a copious inflammatory effusion of serous lymph. The inner aneurism, on the contrary, had a distinct sac, and the smooth structures composing it presented no cognisable traces of recent inflammation. These differences were unmistakeable, and they justify the hope that a smoothly lined aneurism would resent the introduction of a foreign body much less than that which was filled with wire in the present case, or that it might even not be irritated at all.

By these foregoing considerations the prospect of benefit from a repetition of the operation is both pointed out and restricted. Fibrin, it seems, in any quantity, can be attracted by a foreign body inserted into an aneurism, but the result of its accumulation will be inflammation. This result may possibly follow, whether the foreign body which elicits the fibrin from the blood be left within the aneurism or withdrawn from it. In any future operation the object must therefore be to diminish the severity of that process. What inflammation attends the natural deposition of fibrinous strata in an aneurism is always moderate, never dangerous to life, whilst that in the present case was of needless severity. The deposition of fibrin, like many of the great changes effected by disease, is tolerated if not brought about tumultuously; and its deposition by artificial means would also be tolerated if procured in a manner more resembling the natural method, whether

in successive portions or in a smaller quantity on one occasion.

Three modifications of the operation occur to me. Either the quantity of wire employed should be less, or it should be inserted for a certain time and withdrawn again. For the latter purpose it would be desirable to use slender needles, with round, not lancet-shaped, points, instead of the wire and canula. They might be thrust into or across the cavity of the aneurism, the adjoining needles being inserted from opposite sides alternately. They would require to be removed with great gentleness, and all together. It might be hoped that in two or four hours a considerable quantity of fibrin might have collected on the needles, which would on their withdrawal be left in the aneurism. I do not feel competent to speak positively on the consequences of such a proceeding. It might prove more safe than that which I adopted, and may consequently supersede it. In that hope I suggest it, although at the same time I cannot but think it hazardous to presume upon the possibility of the fibrin acquiring in two or four hours a firm adhesion to the aneurismal wall, and hardly less hazardous to make so many punctures as might be needed, were all made at once; while if only a few needles be inserted at a time there would be grave reason to fear the detachment and escape of fragments of the fibrin so soon as the support of the needles was withdrawn.

The second suggestion, which also is drawn from the rapidity with which fibrin appears to have collected in the aneurism, is that it may be possible to effect an equal consolidation with a less quantity of wire. The filling of the cavity with fibrin would take a longer time if the surface exposed for it were less, but the wire would remain to support the soft substance, which would thus be the less liable to break off. Should this method be adopted, it would be important so to manipulate the wire as to ensure its coiling in the cavity, and not merely lying in circles against the wall.

By a third modification of the operation a smaller quan-

tity of wire might be introduced and left in the aneurism; and at the same time, or on successive occasions, needles might be passed in various directions among the coils of wire. After having served to increase the fibrin adhering to the wire, the needles might be withdrawn again.

It must be determined by future experience whether the amount of irritation following the operation would be materially less if, instead of iron, a wire of steel, silver, or gold, were employed.

Postscript, by Mr. Moore, August, 1864.—Since this paper was read, I have become aware that, when making experiments on the possibility of obliterating arteries by transfixing them with needles from the surface of the body, it occurred separately to M. Velpeau and to Mr. Benjamin Phillips, that aneurisms might be treated in a similar manner. M. Velpeau's publication on the subject is in the 'Mémoires de l'Académie des Sciences,' for December, 1830; Mr. Phillips's, in the 'Medical Gazette,' 1831, p. 499, and in a pamphlet, entitled 'A Series of Experiments for the purpose of Showing that Arteries may be Obliterated without Ligature, Compression, or the Knife,' 1832. Although their suggestion was not founded on the principles set forth in the first portion of this paper, and their operation consequently differed from that which I adopted, yet there is sufficient connection between their thoughts and my own on the subject to make it just to refer to their writings upon it.

TWO CASES

OF

STONE IN THE BLADDER OF THE FEMALE,

TREATED BY RAPID URETHRAL DILATATION;

WITH

REMARKS ON THE OPERATION.

BY

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Received Feb. 8th.—Read April 26th, 1864.

HANNAH C—, æt. 52, a fat but healthy married woman, was admitted, under my care, into Guy's Hospital, on October 23rd, 1862.

For eight months she had been suffering severely from vesical irritation, and for the last three she had been unable to hold her urine.

A few days before her admission she had passed two small calculi, but these had, unfortunately, been thrown away.

When admitted she was suffering intensely from bladder symptoms; she was in constant pain, which at times became much aggravated, particularly by walking or maintaining

the erect posture. She had also complete incontinence of urine, which dribbled away as fast as secreted. Her general health was impaired, for she was worn out by pain and want of sleep.

A local examination at once revealed the true cause of all her distress, and the passage of a sound detected the presence of a stone.

There was little doubt, in this case, what was to be done. Lithotrity was necessarily excluded from consideration, on account of the highly inflamed and contracted condition of the bladder, and neither vaginal lithotomy nor urethral seemed to me suitable. The removal of the calculus by urethral dilatation was consequently determined on, and on November 19th the operation was performed. Chloroform was administered, and Weiss's dilator introduced into the urethra; with a few rapid revolutions of the screw the parts were readily dilated, so as to admit my finger; the forceps were then introduced, and the stone seized, but little force being needed for its extraction.

The calculus measured one and a quarter by one inch in diameter, and, with the lithotomy forceps, occupied a considerable space.

The operation was followed by immediate and marked relief. The day following she was able to hold her urine for twenty minutes without pain, and in a week she could hold it for many hours. The urine soon became clear and healthy; and, although her convalescence was retarded by a bronchitic attack, she left the hospital on December 7th, three weeks after the operation, quite well.

Case 2.—Reported by Dr. Beeby.

Ann C—, æt. 85, a married woman, the mother of seven children, was admitted into Guy's Hospital, under the care of Dr. Oldham and Mr. Bryant, on February 17th, 1864.

She stated that she had always enjoyed good health, and that her first symptoms of illness appeared, fifteen months ago, as pain in the loins. At this time, however, her urine

was natural, and she had no bladder symptoms, for these had only made their appearance during the last six or seven months, and had gradually become much worse. When admitted her general aspect was careworn and anxious, her countenance denoting great suffering. She was in constant pain, and unable to retain her urine for more than a quarter of an hour. The act of passing it was both accompanied and followed by severe straining and great pain. The urine was intensely fetid, containing pus and mucus in abundance, with some blood.

The passage of a sound at once detected the presence of a calculus, but any examination caused her great distress. She was kept in bed for two or three weeks, alkalines being administered, and by March 5th her general and local symptoms had so much improved that Mr. Bryant determined to remove the stone.

The patient was first placed upon her back, and brought completely under the influence of chloroform. Weiss's dilator was then introduced, each arm of which had been previously covered with a piece of india-rubber tubing, and with a few rapid revolutions of the screw the urethra and neck of the bladder were readily dilated. The forceps were then inserted, and the stone seized, but little traction being required for its removal. The stone was caught in one of its narrow diameters, but it measured with the forceps exactly two inches.

It weighed, after removal and washing, ℥ij ʒij, and measured five and a quarter by four and a half inches in circumference, and two by one and a half inches in diameter.

It had evidently been resting at the base of the bladder for many weeks, the deposition of the phosphates having only taken place upon its upper surface and circumference.

The operation was attended by the discharge of a little blood, but there was no other point worthy of record beyond the fact that the stone was extracted with great facility, and when passing from the urethra it was rapidly expelled.

The woman passed a good night after the operation, and

the next day she was nearly free from pain; she had recovered the power over her bladder, being able to retain her urine as long as she could before the removal of the stone—a period of fifteen minutes.

On the second day she could hold her water for forty minutes, on the third for two and a half hours, and on the fifth for five hours. The urine also rapidly improved in character. On March 12th, or the sixth day, Mr. Bryant made a careful examination, and found a small slough had separated in the urethra. Into the opening thus made his finger passed, and the neck of the bladder was consequently again dilated. The next day some incontinence of urine reappeared, and it was several days before the patient had recovered control over her bladder.

In one week she could retain her urine for three hours, and on April 5th for five hours; but when walking with a full bladder some little escape took place. Perfect rest was consequently reinforced, and a tonic, in the form of iron, given, the patient, at that time, having full power over her bladder for three or four hours.

Remarks.—The two cases just recorded are good examples of an affection not very common among women; and the complete and rapid recovery after the removal of the stone by urethral dilatation in both instances may well serve as a text to the following remarks, in which I trust to be able to show that the fear of a permanent incontinence of urine—which has been generally associated with the operation of urethral dilatation—is comparatively groundless, and that, within certain limits, the removal of a calculus by *rapid* dilatation is the best and most expeditious practice.

I believe I shall also prove this incontinence of urine to be a common result of urethral lithotomy, and that such an operation should not, therefore, be performed.

Some few remarks on lithotrity in the female will also follow, and the subject of vaginal lithotomy will be touched upon if only to demonstrate the fallacy of the asser-

tion which a great authority is stated to have made—"that it is the only justifiable operation for stone in the female bladder."[1]

On the Dilatability of the Female Urethra.

To prove the great dilatability of the female urethra, it is only necessary for us to recall the details of the few cases which have from time to time been published, in which large vesical calculi have been expelled by natural efforts (see Table A, at the end), and from the early numbers of the 'Philosophical Transactions' some interesting examples may be extracted.

Dr. Molyneux relates an instance in which a stone nearly eight by six inches in circumference was passed from a female æt. 60, "by the help of nature alone."

Dr. Beards relates another in which a calculus of nearly the same size was passed by a woman æt. 63; and he adds, "that it came away with a noise which very much surprised the whole company."

Dr. Leprotti quotes a third, in which a woman æt. 50 passed a stone three and a quarter by one and a half inches in circumference, "its escape being preceded by a discharge of three pints of blood."

Mr. Samuel Cooper, in his 'Surgical Dictionary,' related a case, by a Mr. Middleton, in which a stone four ounces in weight was passed in a fit of coughing, after lodging in the urethra one week; and another by M. Colot, in which a calculus the size of a goose's egg was voided in a paroxysm of pain after a fit of retention of urine.

In all of these instances we are made acquainted with the fact that the calculus was passed; but it is not related whether the bladder regained its natural functions in any, or whether a permanent incontinence of urine was the result, although in all it was added that a recovery followed.

[1] 'Lancet,' Jan. 23rd, 1864. Dr. Marion Sims.

In the following examples the history of the cases is more perfect, and therefore more satisfactory.

In the third volume of the 'Guy's Hospital Reports' a case will be found recorded by Sir A. Cooper, in which a young woman æt. 18, after suffering from symptoms of stone for eight years, passed a calculus two and five eighths inches by one and three eighths of an inch in diameter, "with force, after ten minutes' straining," and *no* incontinence of urine followed.

In the 'Lancet' for January 4th, 1851, a second case will be found, by Mr. Forget, in which a stone three inches long was passed by a woman æt. 50, by a "rapid expulsion," without the slightest evil result; and Dr. Beatty relates an example, in the 'Dublin Quarterly Journal' for May, 1863, in which a woman æt. 40 suddenly passed a stone one and a half by one inch in diameter, accompanied with blood, and no incontinence of urine ensued.

From these cases it is clearly evident that the female urethra may be dilated by natural processes to an extreme degree, and that incontinence of urine need not necessarily be the result.

In the next series I purpose to show the effect of an artificial dilatation of the urethra, or rather the results of a practice founded on the facts which the former cases have furnished.

On the removal of a Calculus by Dilatation of the Female Urethra.

It is always a difficult task to fix the time when any new practice was first introduced; but it can be stated, on the authority of Sir A. Cooper, that it was from the success which followed the practice of Mr. Thomas, in the year 1809, that he was led to dilate the female urethra for the removal of a calculus or foreign body.

Mr. Thomas, however, employed the sponge-tent as the dilating medium; and after its application, which was continued at intervals for three days, he succeeded in dilating

the urethra sufficiently to introduce the forceps, and remove an ivory toothpick from the bladder of his patient.

In the year 1822 Sir A. Cooper had occasion to remove a stone from a female bladder; and, guided by the experience which Mr. Thomas's case had afforded, he recognised the practice of dilating the urethra, adding, "It only remains that it should be considered if better means cannot be devised to produce its dilatation than the introduction of sponge-tents into the urethra."

It was at this date, therefore, that Sir A. Cooper first suggested, and Mr. Weiss devised, the well-known urethral dilator which has been since employed.

Sir A. Cooper, in the twelfth volume of the 'Transactions' of this Society, gives the details of four cases in which this instrument was used with marked and complete success, and in *none* of which did any incontinence of urine result, and states his opinion, "that the greatest advantage of the removal by dilatation over other forms of practice is in the preservation of the powers of retention."

Since that period just mentioned upwards of forty years have elapsed, and during this interval many cases of calculus vesicæ in women have passed under treatment; but it can hardly be stated that there are now any definite rules to guide the surgeon in his treatment of these rare but interesting cases; the fear of a permanent incontinence of urine, after nearly every form of operation, so haunting the mind of the practitioner as to leave him undecided which method to adopt.

Some time since I was, therefore, led to collect all the published cases of stone in the female bladder which I could find, with the view of seeing what light *cumulative* evidence would throw upon this subject, for the experience of any individual surgeon on such a point is necessarily very limited; and as the results are of some interest, I have thought them worthy of being brought before your notice.

In the table marked B, at the end of this paper,

there are tabulated twenty-eight cases of vesical calculus, in all of which the stone was successfully removed by urethral dilatation; and in only four was there any subsequent incontinence of urine, or in about one case in seven.

But if we analyse these cases more carefully, it will be seen that in each of the four unsuccessful cases the urethra had been *slowly* dilated, whilst in most of the twenty-four successful cases the dilatation had been *rapid*.

So far, therefore, as the analysis of the cases which have been tabulated goes, the practice of rapid urethral dilatation appears a sound one.

If we examine the cases more in detail, the same conclusion comes out with equal clearness, for it will be seen that, amongst the completely successful cases, it is recorded that Mr. Simon removed a calculus the size of a gherkin from a woman æt. 35 by rapid dilatation.

Mr. Erichsen took away a stone the size of a shilling from a child three and a half years old.

Mr. Howard extracted from a woman æt. 23, a calculus two and a quarter by one and a half inches in *diameter* with complete success; and Dr. Crisp, from a woman æt. 40, a stone nearly as large, with a like result.

Sir A. Cooper removed from an adult two large calculi, without any unfavorable result.

And in the two cases I have to-day brought before your notice the stones were respectively one and a quarter by one inch in diameter, and two by one and a half inches.

In the remaining cases the calculi were all smaller than those to which I have specially alluded, or were foreign bodies of a smaller diameter.

As a conclusion, therefore, it appears tolerably clear—

That the evidence furnished by the cases of artificial dilatation of the urethra well bears out the conclusions drawn from the former cases in which a calculus had been naturally expelled, and that the following practical deductions may be confidently made:

That the female urethra may be dilated to a considerable

extent with perfect safety, and that rapid dilatation is preferable to slow;

That all small calculi and foreign bodies may be readily removed by this method; and

That in children calculi one inch in diameter, and in adults two inches, may be safely removed by these means.

The operation.—It seems to be advisable that the operation should be performed upon a patient under the influence of chloroform, for the process of dilatation is thus rendered comparatively easy; and it should be rapidly performed. In the two cases I have recorded, this practice was followed, and the dilatation was effected with the greatest ease.

In median lithotomy in the male subject rapid and extreme dilatation of the neck of the bladder has been proved to be not only practicable, but unattended with danger; and, by analogy, therefore, this practice tends to support the one I am now advocating, for most surgeons will admit that elastic tissues recover their tone more readily when rapidly stretched than when subjected to a slow and tedious process of extension.

Weiss's instrument is all that is required for this purpose, although, as originally invented, the edges of the arms, when expanded, are prone to cut the mucous membrane, but, to prevent this taking place in the second case recorded, I adopted the simple expedient of covering each arm with a thin india-rubber coating, this covering fitting into a groove upon the surface of the instrument.

Dilatation by the india-rubber dilator, as employed for obstetrical purposes, or by the modification of Arnott's dilator, as suggested by Mr. Spencer Wells, may also be employed; but I give the preference to the instrument I have just noticed. The surgeon obtains all he requires by its use, and it is very simple.

On the removal of a Calculus by Urethral Lithotomy.

Under the above heading I propose to include all the forms of lithotomy in which the urethra is incised for the removal of a calculus, whether the incision be made upwards or laterally, or whether it be associated or not with some urethral dilatation.

On referring to the analysis of the cases arranged in Table C, it will be seen that out of twenty examples nine were left with some incontinence of urine, or nearly one half of the cases; and when this proportion is compared with that resulting from the analysis of the cases treated by dilatation alone, the result appears still worse, for in the latter, it will be remembered, there were but four instances out of twenty-eight in which incontinence ensued.

If we look to the cases in detail for some explanation of this difference in the results, none can be found beyond that which belongs to the operation itself, for in only one case was the calculus very large.

As a conclusion, it appears tolerably clear that the operation of incising the urethra is one of danger; for incontinence of urine is a very frequent result of such a practice, and, as a consequence, the practice of removing a calculus by such means is not to be recommended.

On the removal of a Calculus by Lithotomy.

It has been already demonstrated with some certainty that the practice of removing a calculus from the female bladder by urethral dilatation is by no means so dangerous as surgeons have been led to believe, and it has been fairly concluded that it is by such means all calculi of moderate, if not of tolerable, dimensions, should be extracted.

It has also been shown that the operation for the removal of a stone by cutting the urethra and neck of the bladder is one of danger, for incontinence of urine is a very frequent result of such a practice.

When we come to consider the results of the removal of a calculus by lithotomy, we have but poor material from which any conclusions can be drawn; although it must be added that the evidence we possess by no means militates against the practice. (See Table D, p. 167.)

In the three cases by Messrs. Fergusson, Birkett, and Cutler, the operation was a simple one, and in all a good recovery was obtained.

In four instances the urethra was also incised, and in one of these some incontinence of urine was left.

In four others the urethra was at the same time dilated, and in one an incontinence of urine was the result, but in this case the dilatation had been prolonged.

It has been already shown, however, for the removal of moderately large stones, that rapid urethral dilatation is a safe and speedy practice, and, as a consequence, it may be fairly assumed that in such cases the operation of lithotrity is not required; but in certain others, nevertheless, it is of great value. When the stone is large and the bladder healthy, lithotrity is, without doubt, the best and most satisfactory operation, the portions of broken stone being subsequently taken away with the lithotomy forceps.

Upon the removal of a Calculus by Vaginal Lithotomy.

It has been reported in one of our periodicals that Dr. Marion Sims, the well-known American surgeon, has expressed a very positive opinion that vaginal lithotomy is the only justifiable operation for the removal of a calculus from the female bladder, and within the last few years several surgeons in this metropolis have revived the practice. Dr. J. H. Aveling, of Sheffield, in an admirable paper which was read before a sister society early last year, has collected from all available sources some very interesting cases in which success was secured by such a practice, but he has failed to prove the truth of the remark made by Dr. Sims.

It is hardly necessary for me to enter more fully into

this subject on the present occasion, for I believe the facts I have brought before you relative to the success which has attended other forms of practice are amply sufficient to prove that vaginal lithotomy is not a necessary operation in the treatment of the majority of cases of stone in the female bladder; although, perhaps, we may endorse the opinion of Dr. Aveling, that, "*when* lithotomy has to be performed in the female, the vaginal method will be the one invariably adopted;" for in cases of very large calculi, in which removal by urethral dilatation is evidently unsuited, and in others, in which lithotrity cannot be performed on account of the condition of the bladder, vaginal lithotomy recommends itself to our consideration.

By way of summary, the following conclusions may be drawn up:

Conclusions.

1. That the female urethra may be dilated to a considerable extent with facility and without danger.

2. That *slow* and tedious dilatation of the urethra by sponge-tents or other means appears to be injurious.

3. That *rapid* urethral dilatation, with the patient under the influence of chloroform, is the safest and more expeditious method of removing all average-sized calculi and foreign bodies from the female bladder; for calculi one inch in diameter in children, and even two inches in adults, have been safely extracted by this practice.

4. That the operation of incising the neck of the bladder and urethra is one of danger, and should be laid aside.

5. That *lithotrity* is a valuable operation in cases in which a stone cannot be safely removed by rapid urethral dilatation, that is, when the stone is large and when the bladder is healthy; and that it is the best practice to remove the broken fragments by the forceps as speedily as possible.

6. That *vaginal lithotomy* is an operation of value when the other modes of operation are inapplicable, but that as a general practice it is not required.

A.—*Cases of Stone in the Bladder of the Female.*—EXPELLED BY NATURAL EFFORTS.

No.	Reference.	Surgeon.	Patient's Name.	Patient's Age.		Size of Calculus.	Incontinence or not.
1	'Guy's Hospital Reports,' 1838.	Sir A. Cooper and Mr. Harris.	M. B.	18	Passed with force, after ten minutes' straining.	2⅝ by 1⅜ of an inch in diameter.	None.
2	'Lancet,' Jan. 4, 1851.	M. Forget.	—	50	Rapid expulsion.	3 inches long.	None.
3	'Medical Gazette,' Feb. 7, 1840.	Roberts.	J. J.	56	Expelled through the vagina by ulceration.	6½ by 6 inches in circumference.	—
4	Ditto, Aug. 24, 1861.	—	—	5½	Rapid expulsion.	1 inch 7 lines by 9 lines in diameter.	Not stated.
5	'Dublin Quarterly,' May, 1863.	Beatty.	E. G.	40	Rapid expulsion, attended with blood.	1½ by 1 inch in diameter.	None.
6	'Cooper's Surgical Dictionary.'	Middleton.	—	Adult.	Expelled in a fit of coughing, after lodging in the urethra for one week.	4 ounces in weight.	—
7	Ditto.	M. Collot.	—	Adult.	Expelled in a paroxysm of pain, after a fit of retention.	Size of goose egg.	—
8	'Philosophical Transactions,' vol. xii.	G. Gardin.	—	—	—	5 by 4 inches in circumference.	—
9	Ditto, vol. xv.	Dr. Wilks.	—	63	—	5½ by 4¾ inches in circumference.	—
10	Ditto, vol. xvii.	Dr. Molyneaux.	—	60	By the help of nature alone.	7⅜ by 5½ inches in circumference.	—
11	Ditto, vol. xxxiv.	Dr. Beard.	—	63	Came away with a noise which very much surprised the whole company.	7½ by 4½ inches in circumference.	—
12	Ditto, vol. xlii.	Dr. Leprotti.	—	50	Escape, preceded by the discharge of three pints of blood.	3¼ by 1½ inches in circumference.	—
13	Ditto, vol. lv.	Dr. Heberden.	—	67	Expelled without much pain.	3½ by 4½ inches in circumference.	—

B.—*Stone in the Bladder of the Female.*—Removal by dilatation.

No.	Reference.	Surgeon.	Patient's Name.	Patient's Age.		Size of Calculus.	Incontinence or not.
1	'Med.-Chir. Transactions,' vol. iii.	Dr. Yelloly.	J. M.	Adult.	*Slow* dilatation.	7¾ by 5½ inches in circumference.	Yes.
2	Ditto, vol. viii.	Sir A. Cooper.	P. K.	Adult.	*Slow* dilatation for 25 hours, by sponge-tents.	1 by ¾ inch in *diameter.*	Yes.
3	'Lancet,' Aug., 1831.	Foster.	Mrs. B.	45	Dilatation for 4 hours.	3¼ by 2½ inches in circumference.	Yes.
4	Ditto, July, 1832.	Brougham.	E. W.	19	Dilatation for 7½ hours.	7 by 6 inches in circumference.	Yes.
5	Ditto, vol. ii, 1855.	Erichsen.	—	3½	Dilatation by sponge-tent.	Size of a shilling.	None.
6	Ditto, 1855.	Howard.	—	23	Dilatation for 2 hours.	2½ by 1½ in. in *diameter.*	None.
7	Ditto, July 26, 1856.	Crisp.	—	40	Dilatation for 2½ hours.	5½ by 3½ inches in circumference.	None.
8	'Guy's Reports,' vol. viii.	Steel.	—	26	Dilatation for 3 days.	Bone bodkin.	None.
9	'Med.-Chir. Transactions,' vol. xii.	Sir A. Cooper.	M. C.	Adult.	Dilatation for 8 hours.	—	None.
10	Ditto, vol. viii.	Ditto.	E. N.	11	Dilatation for 3 days by sponge-tent.	3½ by 3 inches in circumference.	None.
11	Ditto, vol. i.	Thomas.	—	35	Dilatation for 3 days by tent at intervals.	Toothpick.	None.
12	'Cooper's Surgical Dictionary.'	S. Cooper.	—	8	Slow dilatation.	ʒiss in weight.	None after a few days.
13	'Dublin Journal,' Nov., 1863.	Porter.	—	Adult.	Slow, by sponge-tent for 4 days.	Hairpin.	None.

14	'Lancet,' Nov., 1852.	Simon.	—	35	Rapid dilatation.	Size of gherkin.	None.
15	Ditto, Oct., 1857.	Hillman.	—	46	Ditto.	2¾ by 1¾ inches in circumference.	None.
16	Ditto, ditto.	Ditto.	—	5½	Ditto.	2 by 1¾ inches in circumference.	None.
17	'Med. Times and Gazette,' July, 1859.	Le Gros Clark.	—	57	Ditto.	2½ inches in circumference.	None.
18	Ditto.	Moore.	—	30	Ditto.	One ounce in weight.	None.
19	Ditto.	Stanley.	—	41	Ditto.	Size of damson.	None.
20	Ditto.	Teale.	—	48	Ditto.	ʒvj in weight.	None.
21	Ditto.	Cadge.	—	50	Ditto.	—	None.
22	Ditto, 1850.	G. Gwynne.	A. T.	8½	Ditto.	ʒiss in weight.	None.
23	Ditto, 1851.	Davey.	G. B.	3	Ditto.	ʒviss in weight.	None.
24	'Med.-Chir. Transactions,' vol. xii.	Sir A. Cooper.	—	Adult.	Ditto.	Catheter.	None.
25	Ditto.	Chapman.	—	Adult.	Ditto.	Catheter.	None.
26	Ditto.	Sir A. Cooper.	Mrs. B.	45	Ditto.	Two large calculi.	None.
27	—	Bryant.	H. C.	52	Ditto.	1⅞ by 1 inch in *diameter*.	None.
28	—	Bryant.	A. C.	35	Ditto.	2 by 1½ inches in *diameter*; 5½ by 4½ inches in circumference.	None.

13 cases of slow dilatation { 4 with incontinence, 9 without.
15 „ rapid „ all without.

C.—*Stone in the Bladder of the Female.*—REMOVAL AFTER INCISING URETHRA.

No.	Reference.	Surgeon.	Patient's Name.	Patient's Age.		Size of Calculus.	Incontinence or not.
1	'Lancet,' Sept. 13, 1839.	Chevasse.	Mrs.M.	45	Incision upwards.	Pigeon's egg.	Yes.
2	'Lancet, June, 1852.	J. Adams.	—	47	Incision.	—	Partial.
3	'Lancet,' 1853.	B. Cooper.	E. R.	6	Incision on left side.	150 grains.	Yes.
4	'Med. Times and Gazette,' July, 1859.	Ditto.	—	10	Incision.	Small.	Yes.
5	Ditto.	King's College.	—	28	Lateral incision.	Pin as nucleus.	Yes.
6	Ditto.	Bristol Infirmary.	—	10	Incision upwards.	Size of walnut.	Yes.
7	Ditto.	Ditto.	—	15	Ditto.	Size of bantam's egg.	Yes.
8	Ditto.	Shaw.	—	30	Lateral incision.	ʒv in weight.	Yes.
9	Ditto, July, 1859.	Hilton.	—	18	Incision.	Hairpain as nucleus.	Yes.
10	'Lancet,' November, 1852.	Partridge.	J. P.	42	Incision upwards.	2 inches in diameter.	None.
11	Ditto, 1853.	Hawkins.	A. S.	17	Ditto.	Pin as nucleus.	None.
12	Ditto, October, 1857.	Hillman.	—	5	Ditto.	3 by 2¾ inches in circumference.	None.
13	'Med. Times and Gazette,' July, 1859.	Hilton.	—	3½	Lateral incision.	Small.	None.
14	Ditto.	Cock.	—	6	Ditto.	Size of damson.	None.
15	Ditto, 1861.	Stubbs.	E. B.	16	Incision upwards.	ʒvj in weight.	None.
16	Ditto, March, 1860.	Stanley.	—	13	Ditto.	Size of pigeon's egg.	None.
17	Ditto, August, 1860.	Atkins.	—	54	Lateral incision.	6 by 4½ inches in circumference.	None.
18	Ditto, May, 1861.	Stubbs.	E. C.	16	Incision upwards.	ʒv in weight.	None.
19	'Dublin Journal,' vol. xiii.	Steward.	Mrs.G.	50	Incision.	Size of nutmeg.	None.
20	Ditto, vol. xv.	Smyley.	—	76	Incision upwards.	Large oval.	None.
21							

D.—*Stone in the Bladder of the Female.*—Removal by lithotrity.

No.	Reference.	Surgeon.	Patient's Name.	Patient's Age.		Size of Calculus.	Incontinence or not.	
1	'Lancet,' 1853.	Cutler.	S. T.	30	Two operations.	—	None.	—
2	Unpublished.	Birkett.	C.	12	One operation.	(Cystine.) ¾ of an inch in diameter.	None.	—
3	Ditto.	Ditto.	G.	45	Several operations.	Innumerable calculi.	None.	Prolapsus uteri.
4	Ditto.	Perrochaud, of Boulogne.	—	Adult.	Ditto.	Ditto.	None.	Prolapsus uteri.
5	'Lancet,' 1859.	Fergusson.	—	10	Two operations.	—	None.	—
6	'Lancet,' 1852.	Poland.	E. R.	2½	Two operations. Urethra incised upwards.	—	None.	—
7	Ditto, 1852.	J. Adams.	—	7	Ditto. Ditto.	—	None.	—
8	Ditto, 1854.	Fergusson.	E. C.	21	One operation. Urethra incised laterally.	Hairpin as nucleus.	Yes.	—
9	Ditto, 1861.	Hall.	—	Child.	Ditto. Urethra incised upwards.	—	None.	—
10	Ditto, 1852.	Ward.	—	10	One operation after dilating urethra at intervals for 3 weeks.	—	Yes.	—
11	Ditto, 1854.	H. Thompson.	C. T.	9	After dilating urethra for 3 hours one day, and for 4 hours on a second day.	¾ of an inch in diameter.	None.	—
12	'Med. Times and Gazette,' 1859.	Cock.	—	48	Lithotrity, with dilatation.	—	None.	—
13	Ditto.	Humphrey.	—	6	Two operations, with dilatation.	—	None.	—

STATISTICS

OF

QUEEN CHARLOTTE'S LYING-IN HOSPITAL.

BY

GEORGE B. BRODIE, M.D.,

ONE OF THE MEDICAL OFFICERS FOR THE OUT-PATIENTS.

COMMUNICATED BY

CHARLES HAWKINS,

CONSULTING-SURGEON TO QUEEN CHARLOTTE'S HOSPITAL.

Received Feb. 23rd.—Read May 10th, 1864.

A NOTICE having appeared in one of the medical journals for October, 1862, relative to the mortality observed in the lying-in hospitals of London, and in that of Queen Charlotte's Hospital in particular, on my appointment as one of the medical officers to the out-patients of that institution, I, with the consent of Dr. Blakeley Brown and Dr. Mackenzie, the physicians to the in-patients, commenced an examination of the registers, more especially with reference to this subject.

On looking back, I found that the registers had been very carefully kept since the year 1828; prior to 1828 the entries could not be depended on; these, therefore, I have omitted, and have put down in my tables those data only

which may be found recorded; making them, in fact, but an epitome of the practice of the hospital during the last thirty-six years.

Between the years 1828 and 1863 inclusive 7736 patients were delivered in the hospital (producing 7824 children); of these, 3611 were single women, who are admitted on their producing satisfactory proof that it is their first pregnancy; of the 7736 patients, 202 died, 126, or rather less than two thirds of the whole number, being single women.

A great mortality amongst unmarried women on their passage through the puerperal state has always existed. This may be accounted for by their being ashamed of their position, living in retirement for some months prior to their admission, almost, one might say, in a state of solitary confinement, and wanting the common necessaries of life; being separated from their relations and friends, and dreading lest their situation should be found out (this in itself being a considerable anxiety) and forsaken too often by those who effected their ruin. They perhaps possess, at the best of times, but a poor constitution, and in many cases it is to be feared have an improper recourse to medicines; they belong also not unfrequently to a better class of society; they go on hoping against hope till the time of their confinement is close at hand, and then, when the child is born, and there is no escape, overwhelmed with a sense of their own degradation, they sink under an illness from which a married woman would speedily recover.

With regard also to the married patients, many of them, and I refer to the primiparæ, are legally married only within a short time of the termination of their pregnancy; many, I am told, bring their letters of admission to be registered, having been married but a week or two previously; and in one case, which fell under my own observation, the applicant came direct from the church at which she was married to the hospital, registered her letter as a married woman, and was admitted in labour within three days. Under these circumstances, though to all intents and purposes single women, though still obliged to be admitted as married

women, very few constitutions will bear the constant anxiety of "hope deferred" for so many months without evincing in some way or other derangement of function; and when is this so likely to be exaggerated as after the exhausting process of parturition? The mortality amongst the married patients would be therefore considerably diminished if it were not for this fact, that so many of them are exposed during the period of gestation to all the same depressing influences as the unmarried patients.

With these preliminary remarks Table I is introduced; it is designed to show the death-rate for each year since 1828. In the last column of the table is put down the number of women delivered at their own homes annually during this period. The total number of women delivered at their own habitations was 10,858; these, with the 7736 women delivered in the hospital, makes an aggregate of 18,594 patients relieved by the hospital.

The deaths, when added together, yield the following results:—Married, 76; single, 126; excess of single over married, 50. Then, with regard to the death-rate observed in the hospital, a per-centage on the total number of patients delivered will be seen in the table to read thus:

Death-rate, married	1·84
„ single	3·48
Total death-rate . .	2·6

So that the practice of the hospital goes to show that out of every hundred women delivered rather more than two and a half are lost.

From 1828 to 1847 the total death-rate varied from 3·77 to nothing; the three succeeding years, 1848-49-50, show an increased rate, being in 1849 (the year of the cholera) as high as 9·32; from 1851 to 1858 the rate was very low, varying from 1·22 to nil; then, in the three following years, 1859-60-61, it rose considerably, reaching in 1859 as high as 8·12 per cent.; this was in a great measure owing to the increasing number of single women admitted, the mortality amongst whom in these three years was very large.

TABLE I.—*Showing the total number of Patients in each year, with the number of Deaths and the Death-rate.*

Year.	No. of Patients Admitted.			Deaths.			Death Rate.			Patients Delivered at their own homes.
	Married.	Single.	Total.	Marr.	Sing.	Total.	Married.	Single.	Total.	
1828	162	103	265	6	4	10	3·703	3·88	3·77	394
1829	135	86	221	1	5	6	·74	5·81	2·71	379
1830	168	68	236	4	2	6	2·38	2·95	2·54	371
1831	135	72	207	4	...	4	2·96	...	1·93	386
1832	147	70	217	2	..	2	1·36	...	·92	358
1833	95	35	130	...	...	...	...	...	...	334
1834	100	61	161	...	2	2	...	3·27	1·24	286
1835	121	93	214	...	1	1	...	1·07	·46	296
1836	98	71	169	...	2	2	...	2·81	1·18	256
1837	124	91	215	1	1	2	·806	1·09	·93	275
1838	103	99	202	2	3	5	1·94	3·03	2·47	335
1839	108	96	204	2	2	4	1·85	2·08	1·96	327
1840	99	100	199	...	3	3	...	3·	1·5	303
1841	130	88	218	1	2	3	·76	2·27	1·37	331
1842	114	98	212	1	1	2	·87	1·02	·94	329
1843	105	92	197	...	3	3	...	3·26	1·52	306
1844	104	100	204	4	1	5	3·83	1·	2·45	334
[1]1845	73	71	144	1	...	1	1·37	...	·69	265
1846	90	100	190	2	2	4	2·22	2·	2·1	318
1847	96	71	167	3	2	5	3·12	2·81	2·99	266
1848	108	66	174	8	7	15	7·40	10·60	8·62	284
1849	77	84	161	5	10	15	6·49	11·90	9·32	282
1850	88	71	159	...	8	8	...	12·67	5·03	247
1851	82	98	180	...	...	..	...	...	...	312
1852	73	100	173	1	...	1	1·37	...	·57	211
1853	93	84	177	...	...	...	...	...	...	222
1854	129	85	214	...	...	...	...	...	...	190
1855	95	75	170	1	1	2	1·05	1·33	1·18	199
[2]1856	53	35	88	1	...	1	1·88	...	1·13	225
1857	122	123	245	2	1	3	1·64	·81	1·22	236
1858	155	186	341	2	1	3	1·29	·53	·88	230
1859	134	186	320	7	19	26	5·22	10·21	8·12	270
[3]1860	120	136	256	4	14	18	3·33	10·29	7·03	297
1861	160	209	369	3	17	20	1·87	10·13	5·42	350
1862	162	189	351	6	8	14	3·70	4·23	3·92	381
1863	167	219	386	2	4	6	1·19	1·82	1·55	373
Total	4125	3611	7736	76	126	202	1·84	3·48	2·6	10,858

[1] Hospital closed one month for repairs.

[2] New hospital in course of building.

[3] Hospital closed two months for necessary alterations.

TABLE II.—*Showing the total number of Patients in each year at the "Rotundo Hospital," Dublin, with the number of Deaths and the Death-rate.*

Year.	Number of Patients delivered.	Deaths.	Death-rate.
1828	2856	43	1·505
1829	2141	34	1·58
1830	2288	12	·52
1831	2176	12	·55
1832	2242	12	·53
1833	2138	12	·56
1834	2024	34	1·68
1835	1902	34	1·78
1836	1810	36	1·98
1837	1833	24	1·309
1838	2126	45	2·11
1839	1951	25	1·28
1840	1521	26	1·709
1841	2025	23	1·13
1842	2171	21	·96
1843	2188	22	1·005
1844	2176	14	·64
1845	1411	35	2·48
1846	2025	17	·83
1847	1703	47	2·76
1848	1816	35	1·92
1849	2063	38	1·84
1850	1980	15	·75
1851	2070	14	·67
1852	1963	11	·55
1853	1906	17	·89
1854	1943	37	1·90
1855	1060	35	3·30
1856	1600	25	1·56
1857	1509	33	2·18
1858	1084	30	2·76
1859	1389	21	1·51
1860	1404	26	1·85
1861	1135	59	5·19
1862	Not returned.	...	...
1863	"	...	...
Total.....	63,621	924	1·45

The rate, however, is now again decreasing; being in 1862, 3·99; and in the last year, 1·55. I said just now that unmarried women were the cause of the death-rate being so high in certain years; and that this is really so my table shows clearly enough; in one instance only, namely, in 1848, do the married deaths outnumber the single; in 1849 the single were just double the married; in 1850, 8 single and no married woman died; then, again, in 1859, there were the deaths of 19 single to 7 married; in 1860 there were 14 of the former to 4 of the latter; in 1861 there were the deaths of 17 single to only 3 married; these facts show the causes of the high rate of mortality in these six worst years. I need not remark what a much more favorable aspect the death-rate of Queen Charlotte's Hospital would present if its usefulness were restricted to married women.

In another table I have given the death-rate of the Rotundo Hospital at Dublin; and here I must express my obligations to the secretary of that institution for furnishing me with the necessary data. For more readily comparing the relative mortality, I have shown the death-rate annually since 1828 (the year from which the register of Queen Charlotte's Hospital commenced); but as the Rotundo Hospital professes only to admit married women, this death-rate must only be compared with a similar class of patients in Queen Charlotte's Hospital.

In a letter to the 'Lancet' for November 29th, 1862, Dr. Graily Hewitt gives the rate of mortality at the British Lying-in Hospital; he states that for the thirteen years included between 1849 and 1861 there were 1581 deliveries and 11 deaths, being only ·69 per cent. This hospital only admits married women, and those in not larger numbers than about 120 annually; so that, as the editor of the 'Lancet' remarks in a note to the letter, "there is not much scope for the extension of fever."

In the three cases, however, the death-rates stand thus:

Queen Charlotte's Hospital . . .	1·84
Rotundo Hospital	1·45
British Lying-in Hospital . . .	·69

The high death-rate of the Queen Charlotte's Hospital, in my opinion, proceeds in a great measure from a cause that I mentioned at the commencement of this paper, and which I cannot help here repeating, it being a fact of some importance in favour of the hospital; it is, that so many patients come in as married women who have been married only a short time before their confinement, and who have therefore been exposed to all the depressing agencies to which a single woman is exposed during the period of gestation; these patients have hardly a better chance of recovery than the single women who are admitted.

Since the establishment in 1856 of the out-door midwifery department at St. George's Hospital, upwards of 2800 women have been delivered at their own homes, all of them being, according to the rules of the hospital, married. Of the whole of this number 10 died, giving as the rate of mortality ·36 per cent., which, it must be confessed, is very small when compared with 1·84, the rate of mortality at Queen Charlotte's Hospital. Women delivered at their own habitations, as I know by experience, are often living in the greatest filth and poverty, with only one room to accommodate the wants of a whole family, and yet, as will be seen, these patients do infinitely better than those who are removed to a spacious, well-ventilated building, with every comfort and attention that can be devised.

In Table III is shown the "cause of death," arranged according to the frequency of its occurrence; and I may here remark that post-mortem examinations are not the rule at Queen Charlotte's Hospital; they are only performed in cases about which there was any obscurity, or which seemed to call for more minute investigation attending the cause of death. In the table the married patients are divided into primiparæ and multiparæ; the single patients into primiparæ only, for the reason I have given elsewhere.

First on the list comes "puerperal fever," under which

denomination peritonitis, and the other various varieties to which this affection is so prone, must be included. The total number of deaths from this cause is 123, which, being analysed, give to the married primiparæ and multiparæ 41, and to the single patients 82, or three times as many as either of the preceding. The death-rate from this cause reads thus:

Married	·99
Single	2·27
Total rate . . .	1·57

TABLE III.—*To show the Cause of Death.*

Disease.	Married.		Single.	Total.
	Multiparæ.	Primiparæ.	Primiparæ.	
Puerperal Fever . . .	17	24	82	123
Puerperal Mania . . .	1	4	11	16
Phthisis	3	4	11	18
Diarrhœa	...	4	6	10
Post-partem Hæmorrhage .	2	1	2	5
Ruptured Uterus . .	1	1	1	3
Pneumonia	...	...	4	4
Pleurisy	2	...	...	2
Erysipelas	...	1	1	2
Scarlet Fever	...	...	2	2
Measles	...	...	1	1
Disease of the Heart . .	1	...	1	2
Rupture of Aorta . . .	1	...	...	1
Coma following Convulsions .	1	1	1	3
Apoplexy	...	1	2	3
Shock	...	2	...	2
Exhaustion	1	3	1	5
Total . .	30	46	126	202

TABLE IV.—*To show the causes of Death in each year from* 1828 *to* 1863.

Year.	Puerperal Fever.	Puerperal Mania.	Phthisis.	Diarrhœa.	Post-partem Hæmorrhage.	Rupture of Uterus.	Pneumonia.	Pleurisy.	Erysipelas.	Scarlet Fever.	Measles.	Diseased Heart.	Rupture of Aorta.	Coma, following Convulsions.	Apoplexy.	Shock.	Exhaustion.	Total.
1828	9	...	...	...	...	...	...	...	1	...	...	..	...	...	...	...	...	10
1829	3	1	1	...	...	...	..	...	1	...	...	..	...	...	...	...	...	6
1830	4	...	1	...	...	...	...	...	...	...	...	...	...	...	1	...	...	6
1831	2	...	...	...	1	...	...	...	...	...	...	1	...	...	...	...	...	4
1832	2	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2
1833	...	...	...	...	...	...	.	...	...	...	...	...	...	...	...	...	...	...
1834	1	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	2
1835	1	...	...	...	...	...	...	...	...	...	...	.	...	...	...	...	...	1
1836	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	2
1837	...	...	...	1	...	...	...	...	...	...	...	...	...	1	...	...	...	2
1838	3	...	...	...	2	...	...	...	...	...	...	...	...	...	...	...	...	5
1839	2	1	...	1	...	...	...	...	...	...	...	...	...	...	...	...	...	4
1840	1	...	...	...	...	...	...	...	...	...	1	...	...	...	1	...	...	3
1841	...	1	...	...	...	1	...	...	...	...	...	...	...	...	1	...	...	3
1842	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	2
1843	3	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	3
1844	5	...	...	...	...	...	...	...	...	...	...	...	...	...	.	...	...	5
1845	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
1846	2	1	...	...	...	...	...	1	...	...	.	...	...	...	...	...	...	4
1847	5	...	...	...	...	...	..	...	...	...	...	...	...	...	...	...	...	5
1848	12	1	...	...	1	...	...	1	...	...	...	...	...	...	...	.	.	15
1849	14	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1	15
1850	6	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	8
1851	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
1852	...	...	1	...	...	...	...	...	...	...	.	...	...	...	...	...	...	1
1853	...	...	...	...	...	...	.	...	..	...	...	...	...	...	...	...	...	...
1854	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
1855	1	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...	...	2
1856	1	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	1
1857	1	...	1	1	...	...	...	...	...	...	...	...	...	...	...	...	...	3
1858	...	1	2	...	...	...	...	...	..	...	...	...	...	...	...	...	...	3
1859	13	4	2	3	...	...	...	..	...	...	...	1	...	1	...	...	2	26
1860	6	2	4	1	...	...	1	...	...	2	...	...	...	...	...	1	1	18
1861	9	2	4	2	...	1	2	...	...	...	...	...	...	...	...	.	...	20
1862	10	...	1	...	...	...	1	.	...	...	...	...	1	...	...	1	...	14
1863	4	1	...	...	...	...	...	...	...	...	...	...	...	1	...	...	...	6
Total	123	16	18	10	5	3	4	2	2	2	1	2	1	3	3	2	5	202

TABLE V.—*To show the number of Deaths from Puerperal Fever in each month from* 1828 *to* 1863.

Year.	January.	February.	March.	April.	May.	June.	July.	August.	September.	October.	November.	December.	Total.
1828	2	2	2	...	...	1	...	...	...	...	1	1	9
1829	1	...	1	...	...	...	1	...	...	...	...	...	3
1830	...	1	...	1	...	1	...	1	...	...	...	...	4
1831	...	...	...	...	...	...	1	...	1	...	...	...	2
1832	...	...	...	...	...	...	...	...	...	...	...	2	2
1833	...	...	...	...	...	...	...	...	...	...	...	...	...
1834	...	...	...	...	1	...	...	...	...	...	...	...	1
1835	...	...	...	...	...	...	...	...	...	...	1	...	1
1836	...	1	...	...	...	...	...	...	...	...	...	...	1
1837	...	...	...	...	...	...	...	...	...	...	...	...	...
1838	...	...	1	...	1	..	...	...	1	...	...	...	3
1839	...	...	2	...	...	...	...	...	...	...	...	...	2
1840	...	...	...	...	...	...	...	...	...	1	...	...	1
1841	...	...	...	...	...	...	...	...	...	...	...	...	...
1842	...	...	...	...	...	...	...	...	1	...	...	...	1
1843	...	3	...	...	...	...	...	...	...	...	...	...	3
1844	...	...	...	...	...	1	...	...	...	...	1	3	5
1845	...	1	...	...	...	...	...	...	...	...	...	...	1
1846	1	...	...	...	...	...	...	...	...	...	1	...	2
1847	1	...	2	...	1	...	1	...	...	...	...	...	5
1848	2	1	2	...	2	...	2	...	2	1	...	...	12
1849	...	3	4	1	...	...	1	3	...	...	1	1	14
1850	1	...	...	2	...	1	...	...	1	...	1	...	6
1851	...	...	...	...	...	...	...	...	...	...	...	...	...
1852	...	...	...	...	...	...	...	...	...	...	...	...	...
1853	...	...	...	...	...	...	...	...	...	...	...	...	...
1854	...	...	...	...	...	...	...	...	...	...	...	...	...
1855	...	...	...	1	...	...	...	...	...	...	...	...	1
1856	...	...	...	...	...	1	...	...	...	...	...	...	...
1857	...	...	...	...	...	...	...	...	1	...	...	...	1
1858	...	...	...	...	...	...	...	...	...	...	...	...	...
1859	1	1	1	...	1	1	1	...	3	2	2	...	13
1860	1	1	...	...	1	3	...	...	...	...	...	...	6
1861	...	1	2	1	2	...	1	1	...	...	...	1	9
1862	...	1	1	2	2	2	1	...	...	...	...	1	10
1863	...	...	2	...	...	...	1	...	...	...	...	1	4
Total	10	16	20	8	11	11	10	5	10	4	8	10	123

The accompanying Table V, shows in what proportions puerperal fever presented itself in the various months of the year from 1828 to 1863. During eight years, namely, in 1832, '37, '40, '51, '52, '53, '54 and '58, the hospital was free from this scourge of lying-in women.

In 1228, of the 9 deaths, 6 occurred between January and March, 2 to each month; the next case after this happened three months subsequently. It is not my intention to weary the Society with a detailed account of the various dates on which all these fatal cases occurred. I wish to mention those instances only in which any contagion or infection seemed to exist. In December, 1832, there were 2 deaths, the patients being admitted between the 5th and 16th. These two cases were quite isolated. In the middle of March, 1839, there were 2 cases without any apparent exciting cause; these were also quite isolated. In February, 1843, there were 3 deaths, the first of which followed after an instrumental delivery. All these patients were admitted within three days. An interval of fifteen months elapsed before another death occurred.

In November, 1844, there was 1, and in December of the same year there were 3 more cases; in the latter instances the patients were admitted between the 7th and 20th. Between January and July, 1847, there were 5 deaths, 1 occurring in each alternate month, except in March, when there were 2 instances following closely on each other; the first of these was an instrumental delivery. In February and March, 1848, the 3 patients who died were admitted within six days; others also were admitted in the same period, without suffering from the disease; in March the 2 deaths happened within five days of each other, the last death having occurred forty days previously; in May the fatal cases were admitted within five days, and in June on consecutive days; in September, though there were 2 deaths, 1 occurred at the commencement and 1 at the end of the month; there was 1 also in October. After three months' interval no less than 8 cases happened in February, March, and April, 1849. In

February the patients were admitted between the 14th and 26th, and another on the 27th, though this one did not show symptoms of puerperal fever till the following month, March, in which also 3 other cases terminated fatally between the 3rd and 15th. During these months numerous other patients passed safely through the hospital. The next death occurred in July, after an interval of two months, while between the 1st and 5th of August 3 other cases terminated fatally. Of the 2 patients who died in April, 1850, 1 was admitted at the commencement and 1 at the end of the month. In 1859 1 death occurred in each of the early months of the year (except April), up to the end of July; while between September 9th and November 17th, 7 of those admitted died from puerperal fever. On the 7th and 8th of June, 1860, 3 patients were admitted, and they all died from this disease. After an interval of seven months 6 deaths occurred between the middle of February and the middle of May. The last death in this year was in December. In February and March, 1862, there was 1 death in each month; in April the 2 cases were admitted between the 9th and 16th; 4 other cases happened in May and June. The 2 cases in March, 1863, occurred within six days.

From Table III it may be inferred that first labours predispose to the affection. Table V shows that the minimum number of deaths occurred in October and August; next to these, in November and April; that January, July, September, and December, equally rank next; that May and June occupy the next most favorable positions; that February and March are the most unhealthy months; and that 1828, '48, '49, '59, '62, were the most fatal years.

The cause of death standing next on the list is puerperal mania. The numbers here are very striking. Out of the 16 instances of its occurrence only 1 was a multiparous patient, and no less than 11 were single women. The death-rate from mania reads thus:

Married	·12
Single	·304
Total death-rate . .	·206

Nearly all of these were instances of the acute disease, coming on soon after delivery, which, according to Dr. Hunter, is almost always fatal. In 12 out of the 16 cases the age of the patient was between 20 and 24; in the remaining 4 it varied, 1 occurring at 17, the others at 25, 28, and 29 respectively.

Next in the table is phthisis, from which there is a total of 18 deaths; this, again, is by far the most frequently met with amongst the single women, namely, in 11 instances, the remaining 7 being amongst the married patients.

Next in order comes diarrhœa, from which there are 10 deaths, divided between the married and single primiparæ. In a few of these cases the attack came on some time after the patient had been admitted. The larger number were admitted actually suffering from it, or from the exhaustion consequent thereon.

But I will not weary the Society with a detailed account of the remaining cases, which are more or less accidental; suffice it to say that of the 35 patients whose deaths require to be accounted for, 5 died from the effects of post-partem hæmorrhage, 3 from rupture of the uterus, 4 from pneumonia, in all of which old-standing disease of the lungs was present; 2 from pleurisy; 3 cases suddenly, from apoplexy; 3 others after convulsions, with more or less coma; 1 married multipara and 1 single primipara, suddenly, from old-standing disease of the heart; another married woman from rupture of the aorta from fatty degeneration; 2 from erysipelas, from which they were suffering on admission; 2 from scarlet fever, and 1 from measles; 2 from "shock;" and, lastly, 5 from exhaustion. One of these was a twin labour, and 1 was a compound presentation of the "hands and feet."

Lastly, Table VI is intended to show the per-centage of deaths from all causes, in each month of the year:

Month.	No. of Cases per Month, from 1828 to 1863.	Number of Deaths.	Per-centage of each Month.
January	588	19	3·23
February	680	25	3·67
March	800	25	3·12
April	699	15	2·14
May	726	19	2·61
June	702	20	2·84
July	554	19	3·43
August	662	14	2·11
September	579	15	2·59
October	565	7	1·23
November	527	8	1·51
December	552	16	2·9

This has been obtained by comparing the number of deaths from all causes in each month with the total number of patients admitted in the same period, and with the following result:—that the healthiest months were October and November; then August, April, September, May, June, December, March, January, July, and February. This order agrees very nearly with the result of Table IV, having reference to puerperal fever only.

I cannot conclude this portion of my paper more fitly than by giving a short account of the system of ventilation and general arrangement of the hospital. Preliminary to this I would quote the following passages from an address delivered by Mr. Paget before the British Medical Association in 1862. He says—"In every case of erysipelas, pyæmia, or the like, we ought to work till we can discover its probable origin; we should have the strongest feeling that these diseases are not spontaneous nor inevitable. In every case the hospital, or the house, or our own practice, should be brought to trial—to private trial, if you will, yet a just and true trial—a trial before our

own conscience; and if the hospital, the house, or the practice, be found guilty, let it be condemned and amended.

"Of all the remedies I have used or seen in use, I can find but one thing that I can call remedial for the whole disease, pyæmia, and that is a profuse supply of fresh air. In three of the most remarkable recoveries I have seen the patients might be said to have lain day and night in the wind —wind blowing all about their rooms."

Dr. Robert Ferguson, writing on the same subject, says —"Neither the skill, the comfort, the careful dieting, and even the assiduous nursing, which are lavished on the inmates of a hospital, diminish the mortality to a level with that attendant on the out-door population. In her own home the patient is generally better placed, with regard to ventilation, than in most hospitals, and in no malady is pure air, quickly changed, so requisite as in the puerperal state. It is, in fact, the chief prophylactic. A lying-in hospital should consist either of a series of cottages, or its spacious wards should contain very few patients."

"Whatever," says Cruveilhier, "favours the production of hospital gangrene favours that of uterine phlebitis; and, I will add, that both, in their severest forms, are only to be seen in hospitals."

Queen Charlotte's Hospital was founded under the name of the "General Lying-in Hospital," in the Uxbridge Road, in the year 1752. At the end of the last century it was removed to Bayswater, and called "The Queen's Lying-in Hospital;" and in 1809 it was removed to the Manor House of Lisson Green, the present site of the hospital, and since been named "Queen Charlotte's Lying-in Hospital." Many alterations and repairs were made to this building. But it appears that puerperal fever was very prevalent and very fatal on several occasions. Upwards of thirty years ago a ward was erected, detached from the main building, for the reception of patients suffering from this disease.

At last, in 1851, the committee made the following

report on the state of the building:—"That to the want of proper ventilation, drainage, and other sanitary arrangements, they attributed for the most part the prevalence of puerperal fever and the unhealthy state of the hospital." In 1856 the old hospital was entirely removed, and the present one erected. It contains two floors devoted to the reception of patients, one for married and one for single women. On each floor are six wards, containing three beds each, in which the patients are delivered, with an average of 1000 cubic feet space to each patient; but as each ward has not always its complement of three patients, the actual quantity of cubic space to each bed is somewhat increased; on each floor, also, is one convalescent ward containing six beds. The walls of the wards are made of Parian cement, painted, and varnished so as to enable them to be perfectly well washed. Through the building runs a corridor eighty-four feet long by seven wide, having a window at each end, and a large staircase opening at its centre, in the roof of which is a ventilating shaft protected by a cowl. The whole of the hospital is thus completely ventilated.

Each ward is ventilated by means of an opening in a shaft that is carried to the top of the building, an Arnott's ventilator in the chimney, and an opening (that can be closed) over the door communicating with the corridor. The windows are on the plan adopted at St. George's and the Middlesex Hospitals, and form ventilators of themselves.

In each ward there is a constant supply of hot water, and the corridors are heated by the means of pipes containing hot water. The water-closets—one on each floor—are external to the building, and approached through an anteroom, so as to be entirely cut off from the rest of the building. The soil is for many feet of the best gravel, and every attention has been paid to the drainage.

As soon as a patient is able to be moved from the ward in which she has been delivered, she is placed in the convalescent ward, where she remains until she leaves the

hospital. As soon as three patients have been delivered in a ward it is not again used until it has been well cleaned, as well as the beds and bedding. By this plan each ward remains vacant for a week or ten days before patients are again admitted into it. When a case of fever occurs the ward is freshly whitewashed, and the walls thoroughly washed down (in some cases repainted), the bedding purified and re-made, and the ward not occupied again for at least a month. Everything has been done, both in the construction of the building and in the arrangements for the management of the patients, with a view to render them as little liable to disease as possible.

AN ACCOUNT OF A CASE

OF

ANEURISM OF THE ABDOMINAL AORTA,

WHICH WAS CURED BY COMPRESSION OF THAT ARTERY IMMEDIATELY ABOVE THE TUMOUR.

BY

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COMMUNICATED BY

CHARLES H. MOORE, F.R.C.S.

Received April 26th.—Read May 24th, 1864.

History.—The patient (Mark Wilson) is a spare man, twenty-six years old. His occupation as a paviour has required him to use a large wooden rammer for driving paving stones into the ground. Often, in making strenuous efforts, he has overreached himself, and subjected the trunk of his body to severe straining. Eleven months ago, after a hard day's work, he was seized somewhat suddenly with a very severe pain in the back, of a gnawing character, and preventing movement. Two months later the same pain began to be felt in the abdomen, catching his breath, and very severe. He was treated for these pains by several medical attendants, with leeches, blisters, &c. About two months

ago he began to feel a slight pulsation in the belly, and shortly after that time he became my patient at the Newcastle Dispensary, where after a few examinations I became convinced that he had an aneurism of the abdominal aorta. This opinion was shared by the resident medical officer of the dispensary.

The following is the condition of the patient previously to treatment. His abdomen is somewhat spare, so that a distinct pulsation can be seen to the left of and slightly above the umbilicus; the pulsation is most distinct during expiration. On applying the hand, a hard, slightly movable pulsating mass, of a distinct globular form, is to be felt extending from about two inches to the left to about one inch to the right of the umbilicus, and upwards to within three inches of the margin of the left lower ribs. The pulsations in it are very strong, and impinge upon the hand with a sudden stroke, and the expansion of the tumour very distinctly separates the hands when applied to it. The tumour is of about the size of a very large orange. The impulse conveyed to the hand, when laid on the upper part of the tumour, is almost as strong upwards as it is downwards, when felt by the hand applied below the tumour. When pressure is made on the aorta above it all pulsation ceases, and when the pressure is removed a distinct thrill is felt to accompany the rush of blood into the tumour. A line drawn across the abdomen over the umbilicus touches at either end the margins of the last ribs, and encloses between the free borders of the ribs a triangular space (the epigastric region); over the left half of this space there is room enough to compress the aorta against the spine. The aorta below the tumour can be felt, and its pulsations seem in no way to vary from their normal character. By auscultation a feeble bruit can be heard over the tumour. The patient is in good general health; his bowels are subject to occasional attacks of constipation, sometimes being open every day, at others only once in two or three days, which may to a certain extent be accounted for by the large quantity of opiates he has taken to relieve the acute pain which he has suffered.

His arterial system is free from evidence of degeneration.

All palliative treatment having failed to relieve him, after much thought and careful consideration, I proposed to apply a tourniquet above the aneurism, and thus attempt to cure it by compression. It happened, as I have before shown, that between the aneurism and the free borders of the ribs on the left side there was space enough to permit one blade of a tourniquet to press down on the spinal column, and on tightening the tourniquet I found that, by a very nice adaptation, the pulsation in the aneurism could be completely commanded. The instrument used was the ordinary horseshoe tourniquet, made so as to open rather wider than usual, and thus to grasp the trunk of the body.

I took the patient to the Northumberland and Durham Medical Society, to have my diagnosis verified, and to propose my plan of treatment. Several members examined the tumour, and it was admitted by all who did so that the case was unquestionably one of aneurism of the aorta. The president, Dr. Heath, expressed the same opinion in very decided terms. The following is an extract from the 'Report of the Monthly Meeting of the Northumberland and Durham Medical Society, April 14th, 1864:'

"Dr. Murray introduced a patient suffering from aneurism of the abdominal aorta. That it was an aneurism was clear from the fact that when a tourniquet was applied above it pulsation ceased; and on removing the pressure a distinct thrilling rasping sensation was communicated to the hand as the tumour recommenced beating, as if produced by the rush of blood into its interior. As the patient was young, Dr. Murray proposed to operate on the aneurism by pressure. He thought that treatment fully justified, because the disease was otherwise necessarily fatal. Several gentlemen having asked questions and made remarks, Dr. Heath, the president of the society, said "this was a very interesting case; they sometimes met with instances of pulsation of the abdominal aorta, which closely resembled aneurism, but he thought there was no doubt this was truly

a case of aneurism. With respect to the treatment which Dr. Murray proposed to adopt, he thought that since such an eminent surgeon as Sir A. Cooper did once tie the aorta, Dr. Murray was quite justified in employing the milder method of pressure. This was an age of experiment, and he thought the course proposed was a fair and justifiable experiment, and one which possibly might be followed by success."

On Saturday, April 16th, the patient was put under chloroform. (Having lately administered chloroform to a patient for fifteen hours without any apparent injury, I had no hesitation in making a prolonged use of it in this case.) The anæsthetic influence was accordingly kept up for two hours, during which time, except during momentary displacements of the instrument, the pulsation in the aneurism and in the vessels of the lower limbs was completely arrested. On removing the pressure no visible effect had been produced; but he passed no urine for nearly thirty hours. It was found exceedingly difficult to keep up steady pressure, as the patient, being under chloroform, unconsciously moved about a good deal; the irregular action of the muscles of expiration in the abdominal wall added very much to the difficulty, and it became necessary to sit constantly with one hand on the screw of the tourniquet and the other on its anterior or applied blade, and thus to secure the constant pressure required. On neither occasion when the pressure was applied did we escape a considerable number of recurrences of the pulsation from displacements of the instrument. This statement, however, does not apply to the last hour of the second and successful attempt, during which all movement and pulsation were completely arrested.

On Tuesday, April 19th, after much entreaty on my part, the patient again submitted to be put under chloroform. Dr. Heath having carefully re-examined the tumour, and expressed his conviction of the nature of the disease, the tourniquet was applied, and pulsation in the tumour completely arrested. With the assistance of Drs. Nesham and Spencer, and Messrs. Armstrong and Powell, the pressure

and the insensibility were kept up for about five hours. Until the last hour the slightest movement in the tourniquet showed that pulsation in the tumour had not ceased, and that the disease was unaltered. During the last hour the existence of pulsation became less obvious. It was then deemed prudent, lest the patient's strength should be exhausted, to remove the pressure and see what had been accomplished. On finally removing the pressure very slight pulsations were felt, and hopes were entertained that some advantage had been gained. As the femorals did not beat during the application of pressure, the extremities had become cold; and when the patient recovered from the chloroform a fit of shivering occurred. Hot bottles were applied to the feet, and hot brandy and water was given, followed in a short time by a large dose of a mixture of chlorodyne, opium, and belladonna, containing about ʒiss of the tincture of the last drug, which I find is a perfectly safe dose as an anodyne when combined with an ordinary dose of opium. In the evening he was found restless, and "sore all over," with tenderness at the seat of the aneurism and of the pressure, and numbness of the extremities. To my astonishment the tumour was perfectly pulseless, and every indication of pulsation in the aorta below it had disappeared.

Wednesday, April 20th.—Patient feels restless and slightly feverish, with thirst and hot skin, but the pulse is only 72 and feeble. Bowels open, passes water freely; can stand, although the legs are still numb, and he feels "pins and needles" in his feet. In consultation with Dr. Heath, a most careful examination was made, and the following observations were confirmed by that gentleman. There is no pulsation in the tumour, which is now perfectly stationary, hard, resistant, and lessened in size. Nor are any pulsations to be felt in the aorta below the tumour, in the iliacs, or in the femoral arteries.

Thursday, April 21st.—Patient looks well and feels much better; says he is more free from pain than he has been for several months. There is a very slight movement in

the tumour, which is now a hard globular mass, easily felt, and slightly movable, but evidently smaller than before. At one or two points on the abdominal wall pulsating vessels can be felt, but there is no pulsation in the femorals.

Friday, 22nd.—With Mr. Lightfoot, who carefully examined the case, the following points were made out and verified by that gentleman:—A solid hard tumour, of about the size of an apple, lying to the left of the umbilicus, can be felt, and during deep expiration can be seen. It is motionless to the eye, and by the hand the slightest possible forward movement can be distinguished at its upper border, as if communicated from the aorta pulsating above. No expansion, thrill, or bruit, can be made out. Running over the right border of the tumour a vessel can be felt pulsating, which, from its position and size, is probably the superior mesenteric artery. The femorals are pulseless. All numbness is gone from the legs, and the patient declares he feels quite well.

Saturday, 23rd.—Observation of the tumour corresponds with the notes of yesterday in every particular. The pulsation of small arteries in the abdominal wall is now pretty distinct. The patient is sitting up and out of bed, feels better than he has done for months past, and is free from pain. Eats well and sleeps well.

Sunday, 24th.—Going on well.

Monday, 25th.—Still improving, and is moving about freely. The tumour is now much diminished in size, and no pulsation can be distinguished. (Observation confirmed by Dr. Spencer.)

Tuesday, 26th.—The patient has been out this morning, and walked about a quarter of a mile. On ceasing to walk he felt as if a cord was tied around his waist, and was quite numb below that level. He feels his legs numb and weak, but in other respects is quite well and in good spirits, declaring himself to be better than he has been "for eleven months past." After careful examinations, the observations previously made were this morning confirmed by Messrs. Fife and Armstrong. Mr. Rayne, after a very

careful examination, also expressed his conviction "that there is now no blood passing through the tumour." Dr. Gibb and many other medical gentlemen in the town, after examining the parts, came to a similar conclusion.

May 1st.—Patient still improving. Legs warm and stronger, but still numb when he walks far. Had an attack of diarrhœa, which ceased on taking a few doses of chalk mixture. No pulsation in the tumour or arteries below it.

5th.—Still improving. Took a long walk two days ago, and, except a feeling of numbness and weakness in the legs, was no worse for it. Tumour carefully examined this morning in the presence of several medical men, when the following points were observed and verified:—"The tumour is stationary, harder than before, and lessened in size. Its periphery lies seven inches from the sternum (the patient having a long chest) and five inches above the pubes, four inches from the anterior superior spine of ilium on the left side, and five inches from the same point on the right side. It lies a little more to the left than to the right of the umbilicus." No bruit can be heard, and the aorta above can be felt beating in the epigastrium.

11th.—Patient still improving, tumour pulseless, and diminishing in size.

Remarks.—I need offer little comment on this case, especially as the patient will be introduced at the meeting of the Society. I would note, in the first place, that we have in it a most complete triumph for the advocates of "compression" in the treatment of aneurism. The disease, though one which has baffled all attempts to cure it, yet in this instance disappeared by means of treatment lasting but a few hours, and involving the use of a known and simple expedient.

Secondly, here is actual proof that a sudden occlusion of the aorta can take place without violent symptoms or great inconvenience ensuing.

Again, the case shows that in compression, as has been

noted by an eminent surgeon in the north, "the actual cure takes place very rapidly, probably in less than an hour;" for, till within the last hour of the treatment, the slightest movement in the tourniquet was followed by most violent beating in the aneurism.

As an instance of the dependence of a curative process on the influence of chloroform, this case is most striking; for no man, exhausted with pain and weary of life, could have borne for five hours, without an anæsthetic, such tremendous pressure as was here employed, even though that pressure were to save his life.

Postscript, July 7th, 1864.—The patient has obtained a situation as a "fitter," and feels equal to the work. The only unfavorable symptom now present is a numb pain in the knees after walking, which disappears after resting a few minutes. The lower limbs are plump, but flabby; the rest of the body is well nourished. The tumour is now scarcely to be felt, and the aorta, iliacs, and femoral arteries, are still quite pulseless.

Postscript, September 26th, 1864.—I have seen the patient this morning, and he looks well. He only complains of slight weakness and pain in his back and legs. The numbness no longer exists. He works as an engine-fitter from 6 a.m. till 8, and sometimes 10 p.m.; in addition to which he has to walk nearly two miles to and from his work. The abdomen and limbs are now plump and fat. Only slight hardness, which is evident on *deep pressure only*, is perceptible at the site of the aneurismal tumour. There is no pulsation in the aorta below the site of the tumour, but above it a very distinct thud can be felt to strike dead against the applied hand. There is now distinct pulsation in the right femoral artery, but no certain evidence of it in the left. The patient says "he now feels as well as ever he did in his life." Five months have now elapsed since pulsation was felt in the tumour.

CASE

OF

CONGENITAL IMPERFECTION

OF THE

MAMMÆ, SEXUAL ORGANS, STERNUM, AND HEART,

IN A WOMAN AGED TWENTY-TWO YEARS.

BY

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Received April 7th.—Read May 24th, 1864.

THE following case is of so rare and remarkable a character, that I venture to submit it to the Society in its present necessarily imperfect form, although I am fully aware that there are several points of interest connected with it which could only be determined by a post-mortem examination.

H. D—, æt. 22, an unmarried female servant, was admitted as an out-patient at the Middlesex Hospital, under my care, on March 11th, 1864. She was brought to the hospital by her mistress on account of a noisy cough, unattended by expectoration, which, together with a nervous manner and a tendency to cry when spoken to, gave the case at first the appearance of being one of hysteria. She

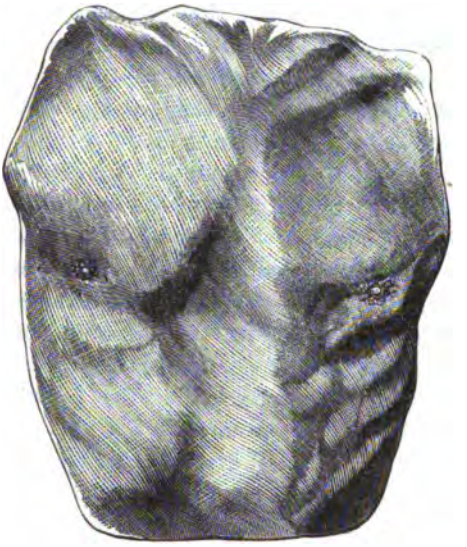
stated that till lately she had been strong and well able to work as a servant, but was now less equal to exertion. Had had no serious illness, and had never suffered from rheumatism in any form. Had been troubled with cough for about a year, and also with palpitation of the heart, breathlessness in going upstairs, or even in quick walking on level ground, and with pain in the left side immediately below the usual situation of the left breast. Had been in a slighter degree subject to these latter symptoms so long as she remembered, and had frequently been affected with cough on exposure to cold, but had become decidedly worse in all these respects since she had been employed as a servant of all work, with much going up and down stairs.

She had never menstruated, neither had she had any feelings of fulness in the pelvic region, any lumbar uneasiness or pain, or any vicarious hæmorrhage, such as hæmoptysis or hæmatemesis.

Is of medium height and of feminine but rather girlish appearance. Complexion clear and florid; voice feminine. Is of spare habit; hair brown and in fair quantity. Eyes brown, general aspect healthy, but hands and forearms midway up to the elbows cold and red, with a slight purplish tint; feet also generally cold. Lips of normal colour, tongue clean. Pulse, in the erect position, variable in frequency, irregular, and exceedingly small; on one occasion it was only 66, but more commonly from 80 to 86; it always increased in frequency, and became quite regular and of better volume, when the patient assumed the recumbent posture, and again fell more or less in frequency, remaining quite regular, after she had lain still for some time. This was verified on several different occasions.

The upper part of the chest is normal, but rather flat. Nearly in a line with the interspace between the second and third ribs a depression commences in the sternum, which is at top about an inch in breadth, but widens and deepens lower down, until over the lower part of the sternum and at the ensiform cartilage it is fully two inches in breadth, and about three quarters of an inch in depth.

The lower costal cartilages appear to be less firm than usual, and yield readily under a moderate degree of pressure. About the middle of the ensiform cartilage a small space, that can be covered by the point of the finger, is exquisitely tender on the slightest pressure, and is stated by the patient to have been so from her earliest remembrance. On examination this tender spot does not, either to sight or touch, present any peculiarity as compared with the surrounding surface, but again and again on different occasions she shrank and complained of pain when the finger was either intentionally or inadvertently pressed on it. The patient has always been aware of the peculiar conformation of her chest, upon which she has never either exerted any undue pressure or received any blow. The chest is perfectly flat in the mammary region, and on the most careful manipulation no trace of the mammary glands could be discovered.



The above woodcut is taken from a photograph of a plaster cast of the chest.

There is a rudimentary nipple in the usual situation on either side; this consists of a small pink-coloured, lens-

shaped elevation, about a line and a half in diameter, and raised about half a line above the surrounding surface. Around each nipple is a very pale pink-brown areola, about two thirds of an inch in diameter, dotted with minute rudimentary follicles, but, instead of projecting a little from the chest, these areolæ are slightly depressed below the surrounding surface.

The chest expands equally on both sides; the resonance on percussion over the lungs is normal; and on examination of the lungs there is no evidence of definite pulmonary disease, but the respiration is dry, puerile, and rather harsh throughout both lungs. The præcordial dulness is increased in extent, especially towards the right side, and the impulse of the heart is forcible and very perceptible to the eye over a considerable space at the lower end of the sternum and beneath the left costal cartilages. The heart's action is regular, and the sounds are clear and free from murmur when the patient is in the recumbent posture, but it becomes at once irregular when she sits or stands upright. A triple sound is then also sometimes heard; the third sound, which appears to be a reduplication of the systole, is always of a murmurish character, and is frequently supplanted by a loud murmur intermediate between the systole and diastole. This murmur is most intense a little below the fourth costal cartilage, but may be heard from the third costal cartilage downwards, and also, though much less loud, in the direction of the axilla. Sounds at the base of the heart both clear.

The hips are less rounded and the pelvis is evidently smaller than is usual in ordinary women of her age. The circumference round the hips is thirty and one eighth inches, that round the shoulders on a level with the acromion processes is thirty-four and a half inches. The same measurements taken in two other women for the purpose of comparison appear to show that these dimensions are abnormal and disproportionate. In a young woman aged eighteen years the measurement round the shoulders was thirty-two and a half inches, that round the hips thirty-one and a half

inches. In the other case, that of a married woman over thirty years of age, who had had one child, and was not pregnant, when measured the dimensions were thirty-nine inches round the hips, and thirty-six and a quarter inches round the shoulders. Thinking it desirable to ascertain the condition of the genital organs, I requested my friend and colleague Dr. Hall Davis to examine the patient, and I subjoin the report with which he has been kind enough to furnish me:

"Mons veneris very slightly prominent, has very scanty covering of hair; skin very fair on these parts. Clitoris and nymphæ present and of normal size. Hymen and vestibule very vascular; the opening through it admits with difficulty the index finger. Vagina very narrow, admits two and three quarter inches of index finger. No os uteri found projecting into vagina, although there is one spot the size of top of index finger which feels thickened. On pressing firmly against the vaginal cul-de-sac no cervix of the uterus, nor anything like ovaries or other solid matter, can be felt beyond it. No orifice can be detected by the touch at the thickened point. No examination by speculum was made, as the vagina was much too narrow for such a mode of inquiry. On examination per rectum there appears to be a uterus, that is to say, a solid body is felt on pressing against the anterior wall of the rectum, but this body is smaller and much less distinct than an ordinary uterus, and nothing resembling ovaries could be discovered."

After a careful search I have only been able to find four cases on record in any respects similar to the above, two of them described by English and two by French authors. Rokitansky, indeed, speaks of the ovaries, together with the other portions of the female sexual organs, as being often found in a state of imperfect development, and says that in such cases the mammæ are likewise found imperfectly deve-

loped.[1] But such cases must nevertheless be rare, or they would have been more frequently described. It will be observed, moreover, that although the four cases about to be quoted all resemble the one now submitted for consideration in the important feature of the undeveloped state of the mammæ, yet they all, except the second case, which closely resembles mine, differ from it in various other respects.

The first English case I have been able to find was communicated to the Royal Society by Mr. Charles Pears in 1805.[2] It was that of a woman named Ann Joseph, who died of pulmonary disease in the twenty-ninth year of her age. She was of a fair florid complexion, with dark-brown hair, and was strong and able to endure hard work. "Having ceased to grow at ten years of age, she was in stature not more than four feet six inches high. Her breadth across the shoulders was as much as fourteen inches, but her pelvis (contrary to what is usually observed in the proportions of the female skeleton) measured only nine inches from the ossa ilia across the sacrum. Her breasts and nipples never enlarged more than in the male subject; she never menstruated; there was no appearance of hair on the pubes, nor were there any indications of puberty, either in mind or body, even at twenty-nine years of age." On post-mortem examination the following appearances were observed in the female organs:—"The os tincæ and uterus had the usual form, but had never increased beyond their size in the infant state; the passage into the uterus through the cervix was oblique. The cavity of the uterus was of the common shape, and the Fallopian tubes were pervious to the fimbriæ; the coats of the uterus were membranous. The ovaria were so indistinct as rather to show the rudiments which ought to have formed them than any part of their natural structure."

[1] Rokitansky's 'Manual of Pathological Anatomy' (Dr. Sieveking's translation for Sydenham Society), vol. ii, pp. 327 and 329.

[2] 'Philosophical Transactions of the Royal Society,' A.D. 1805, pp. 225-7.

The only other English case I have found was described by Mr. Baynham in the third volume of 'The London Medical Gazette.'[1] It was that of an unmarried woman, called Elizabeth Fomes, who died of diseased heart in April, 1829, aged forty-eight years. She had always been delicate, and had never menstruated nor suffered any inconvenience from this deviation from nature. At the post-mortem examination "the uterus was found to be accurately formed, but not in the least developed from the period of infancy, the body of the organ not being larger than an almond. The orifice was well marked and the cavity free. The Fallopian tubes were delicate, but disproportionately long, and their fimbriated extremities beautifully disposed. The ovaries could not be accurately distinguished, being in appearance little else than accumulated cellular substance. The vagina was not particularly short or diminutive. Two other points deserving notice were that there had never been any hair upon the pubes, nor any perceptible formation of the mammæ."

The two French cases were both published in the second volume of the 'Mémoires de la Société Médicale d'Emulation.'[2] They were both, like my own, examined only during life. The first, that of a young woman aged twenty-one years, was described by M. Cailliot; the second, that of a woman aged twenty-five years, by M. Rénauldin. In both cases the vagina consisted only of an extremely narrow canal, and neither of them had menstruated. The subject of M. Cailliot's report had enjoyed uninterrupted good health, and had experienced none of the discomforts and ailments, such as vicarious hæmorrhage, which so frequently attend the absence of the catamenia. The subject of M. Rénauldin's case had begun about the age of puberty to suffer from headache, palpitation, nausea, and occasional vomiting of black matters; and it was on account of these ailments that she had sought medical aid, and had

[1] 'London Medical Gazette,' vol. iii, p. 732.

[2] 'Mémoires de la Société Médicale d'Emulation,' vol. ii, pp. 270-6. Paris, 1798.

even undergone a fruitless surgical operation. M. Cailliot says of his patient that the breasts were but slightly developed, resembling in size those of a girl below marriageable age. In M. Rénauldin's patient the mammæ were entirely undeveloped, not exceeding in size those of an ordinary man.

In the absence of any conclusive examination of the internal sexual organs, the two principal points of interest presented by the above case are the abnormal state of the mammæ and of the heart. Both are the subjects of congenital imperfection, the result probably of arrested development. Regarding the precise nature of the cardiac imperfection, I feel unable to offer any positive opinion; but it seems almost certain, from the result of Dr. Hall Davis's examination, and from the light thrown on the case by the post-mortem examinations quoted above, that the defective development of the mammæ is associated with a rudimentary and imperfect formation of the ovaries.

PATHOLOGICAL RESEARCHES

INTO THE

DISEASES OF THE EAR.

(SUPPLEMENT TO THE SEVENTH SERIES.)

SEBACEOUS TUMOURS IN THE EXTERNAL AUDITORY MEATUS.

BY

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Received March 8th.—Read June 14th, 1864.

In the forty-fourth volume of the 'Medico-Chirurgical Transactions' I gave an account of *eighteen* cases wherein sebaceous tumours, developed in the external auditory meatus, had caused absorption of the petrous bone, and in several of these cases caries had also occurred. In one of the cases alluded to, an abscess in the cerebrum, and in another, one in the cerebellum, had taken place.

Since the publication of the paper referred to, two cases of caries of the temporal bone from the presence of sebaceous tumours, and both of which proved fatal, have fallen under my notice; I therefore think it advisable to place them also before this Society. I hope by doing so I

may induce some of its Fellows to give attention to this very singular affection. It appears that sebaceous tumours may be developed in the substance of the dermoid meatus at any period of life, and they appear always to have the peculiarity of advancing inwards, and causing absorption of the petrous bone. The reason for their doing so is by no means obvious, inasmuch as there appears to be little or no perceptible impediment to their progress towards the free surface of the meatus, and to their extrusion through this canal. Not uncommonly, as in the two cases about to be detailed, the tumours make their way directly into the tympanic cavity, which they completely fill. The fact that the petrous bone is perforated by the pressure exercised by these soft tumours, formed merely of layers of epidermis, is another well-marked example of absorption occurring in the hardest textures when subject to continuous, though gentle, pressure. A fact worthy of notice is that the pressure of the tumour on the external meatus always causes simple absorption of the bony walls, whereas in the tympanic cavity its effect is to induce caries; and the irritation within the tympanum may also give rise to an abscess in the cerebrum or the cerebellum (according to the part of the tympanic cavity implicated), without the existence of any direct continuity of diseased structure from the tympanum to the brain. The peculiarly insidious manner in which sebaceous tumours make progress, often, indeed, scarcely betraying their existence until the appearance of severe, frequently fatal, cerebral symptoms, demands careful consideration, and should lead the medical man to make a thorough examination of the ear even when the slightest symptoms of irritation in the meatus are the cause of complaint. In the two following cases it would appear that the attention of the surgeon had not been called to the state of the ear until the supervention of the symptoms which ended fatally.

For the notes of the following case, and for the opportunity of dissecting the petrous bone, I am indebted to my

colleague Dr. Chambers, under whose care it occurred in St. Mary's Hospital.

CASE 19.—*Sebaceous tumour distending the tympanic cavity; caries of the petrous bone; death.*

N. F—, a girl æt. 16, was admitted into the hospital on December 21st, 1862; she had been ill two days with pain in her neck; she was unable to walk, and she kept her head thrown back. She went through an attack of scarlet fever seven months previously, but perfectly recovered, and was in service till within a few days of her admission. There was no history of deafness or discharge from either ear, nor was any discharge observed when the nurse washed her. Next day she seemed much better, and lay with her head naturally on the pillow. On the third day after admission she complained much of headache, started up in bed several times during the morning, but ate her dinner at one. Half an hour afterwards she suddenly died.

Autopsy.—The tentorium on the left side adjacent to the petrous bone was inflamed, and pus was effused upon it. The pia mater covering that part of the cerebellum beneath the inflamed tentorium was opaque, and a portion of the cerebellum itself, as large as a pea, was softened and of a green colour.

Dissection of the temporal bone.—The dura mater covering the posterior and upper part of the petrous bone was dark coloured and softer than natural, and the bone which it covered contained numerous small apertures. These apertures were so numerous and large in the sulcus lateralis, which forms the posterior boundary to the mastoid cells, that it assumed a carious aspect. The lateral sinus adhered less firmly than natural to the sulcus lateralis, and it contained a dark clot. *External meatus.*—At the upper and inner part, directly above the membrana tympani, was a soft yellowish mass of matter, composed principally of laminæ of epidermis; its removal disclosed a circular aperture in the upper osseous wall of the meatus, about a quarter of an inch

in diameter, its margins being well defined, and the bone around having a healthy structure. This aperture was also filled with a mass of epidermoid laminæ, and it communicated with the cavity of the tympanum. The membrana tympani presented two apertures, one anteriorly and inferiorly, the other posteriorly and inferiorly.

For the particulars of the next case, and for the opportunity of dissecting the diseased parts, I have to thank my friend Mr. Bullock, of Isleworth.

CASE 20.—*A sebaceous tumour in the external meatus, causing caries of the petrous bone, suppuration of the pia mater and death.*

A boy æt. 16, thin and not healthy looking, who was training for an army bandsman, was sent into hospital on February 7th, 1864, complaining of pain in and about his right ear; this he attributed to his having worn a damp cap a few days previously; there was no discharge from the ear, nor, indeed, any abnormal external appearance. A dose of calomel was administered, followed by a purgative, and, the pain continuing, twelve leeches were applied to the region of the mastoid process; a blister was also applied to the nape of the neck; in spite of this treatment, he rapidly became delirious. A week after admission an abscess was opened behind and below the mastoid process, and a large quantity of very offensive pus was discharged. The severity of the cerebral symptoms increased, nevertheless, and the patient died on the eighth day after admission.

Autopsy, three hours after death.—The brain was generally very vascular, and serum was effused in the ventricles. The outer surface of the middle lobe of the cerebrum was intensely congested, and the larger vessels contained coagula; the entire surface of the right hemisphere of the cerebellum was coated with fetid, green, purulent lymph, and the discoloration extended nearly half an inch into its substance. No abscess was detected either in the cerebum or in the

cerebellum, the structure of the brain, with the exception just named, being apparently healthy. The dura mater was detached from the upper surface of the petrous bone, and the bone presented carious orifices. The dura mater over the posterior and outer surface of the petrous bone, and that forming the lateral sinus, was also separated from the bone by offensive pus, and there was also purulent lymph in the lateral sinus itself, together with a considerable coagulum. There was a considerable quantity of serum in the pericardium, purulent serum and lymph in both pleural cavities, and purulent deposits in both lungs.

Dissection of the temporal bone.—At the upper and inner part of the external meatus, close to the *membrana tympani*, was a mass of soft sebaceous matter, which extended, by means of an aperture nearly half an inch in diameter (formed partly in the upper part of the membrana tympani, and partly in the adjacent bony meatus), into the tympanic cavity, which it completely distended. There were small carious orifices in the upper wall of the tympanum, and also in the sulcus lateralis, which contained dark and fetid purulent matter.

ON THE CONDITION

OF THE

STOMACH AND INTESTINES IN SCARLATINA.

BY

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Received April 22nd.—Read June 28th, 1864.

In the following paper I shall attempt to show, that inflammation of the œsophagus, stomach, and intestines, usually accompanies scarlatina; that, desquamation of the epithelium of these parts takes place; that, notwithstanding the anatomical changes in the stomach, the formation of pepsine is not prevented; and that, in this disorder, the condition of the mucous membrane is similar to the condition of the skin.

I.—*That inflammation of the œsophagus, stomach, and intestines usually accompanies scarlatina.*

Observation 1.—A child two years old was suddenly attacked by vomiting and diarrhœa. After a few hours a slight scarlet rash appeared on the body; five hours from

the commencement of the illness convulsions came on, and the child died fourteen hours afterwards.

Post-mortem examination.—The mucous membrane of the stomach was in one part much congested, but did not present to the naked eye any other appearance of disease. Its tubes could be separated with greater ease than usual. When examined by the microscope they appeared large and much distended at their closed ends, only a few scattered cells being visible in them, and their basement membrane seemed everywhere lined by a false membrane (see Plate V). After the stomach had been soaked in spirit for six months a considerable change took place. The tubes then seemed filled with cells and with granular matters agglutinated into masses; but the cells were very much less numerous than in the natural condition. Each cell seemed covered with a thin membrane, which could be seen to pass from one to another. The villi of the duodenum were stripped of epithelium, and, although there was no trace of food in the intestines, the villi were distended with oily and granular matters. The lacteals were empty.

Observation 2.—The stomach of a female child, aged twelve years, who had died of scarlatina, was sent me for examination. The tubes were greatly distended and easily separated from each other. In many places they were stripped of their cells, and contained only granular matters, but in other parts there were large and very thin cells in the tubes. The connection between the tubes and their contents was much less than usual, for when sections were made obliquely the tubes were generally found empty. When the cells were removed from the tubes they appeared fringed with pieces of fine membrane.

Observation 3.—The stomach of a female child who had died of scarlatina at the age of two years was sent to me for examination. The tubes were easily separated from each other, and greatly distended by granular and fatty matters. There were but few gastric cells visible, and in

other respects the appearances were the same as in the preceding case.

Observation 4.—A boy died of scarlatina in the Middlesex Hospital.

Post-mortem examination.—The pharynx and œsophagus were red, and the epithelium was absent. The stomach was covered by a thick tenacious mucus, and when this was removed the mucous membrane was found greatly congested, and in many places presented a blood-stained surface. The intestines were in a similar condition. Peyer's patches were large and congested, and the glands of the mesentery enlarged and of a deep-red colour, as in typhoid fever. The tubes of the stomach were amazingly distended, and their closed ends curved and distorted. After being soaked in alcohol there were spaces left between the basement membrane and their contents. The basement membrane seemed much thickened, and the tubes were gorged with a confused mass of cells and granular matters. Scattered everywhere in apposition with the inner surface of the tubes were small round cells $\frac{1}{4000}$ part of an inch in diameter. There were also a few larger cells $\frac{1}{1100}$ of an inch, very thin and flat, and agglutinated to the rest of the contents. In other specimens no large cells were to be discovered, and in another part the tubes themselves were found united together, the intertubular spaces being loaded with cells and nuclei. The intestinal villi were also loaded with fatty and granular matters, although the lacteals were not distended.

Observation 5.—A girl aged fourteen was admitted into the Middlesex Hospital. She had been affected the night before with sore throat, followed by a slight rash upon the skin. She walked to the hospital in the morning, but was attacked by diarrhœa, and died the same evening.

Post-mortem examination.—The œsophagus immediately above the stomach was very red. The stomach was much injected in the cardiac region, and for some distance from it. There were two or three ulcerations in the solitary glands

of the intestines. The muscular coats and the subtubular and intertubular spaces of the stomach, were in many parts infiltrated with blood. The tubes were distended and the edges of the cells ill-defined, from being overlaid with granular matters. In the pyloric region were deposits of nuclei encroaching on the tubes; these deposits, no doubt, were the remains of some former attack of inflammation of this part.

Observation 6.—A youth æt. 17 died of scarlatina at the Middlesex Hospital, after a few days' illness.

Post-mortem examination.—The stomach was congested at the pyloric region, but there was no unusual redness in other parts of the organ. In the cardiac and middle regions the tubes were distended with round or oval nucleated cells, about $\frac{1}{4000}$ of an inch in diameter, intermixed with granules and a few gastric cells, some of these being granular and larger than usual. Between and below the tubes were numerous cells and nuclei. In the pyloric region the tubes were greatly distended, and masses of granules and small epithelial cells projected from their free ends. They did not appear to contain any normal gastric cells.

Observation 7.—A man was admitted into the Middlesex Hospital, and died a few days afterwards from scarlatina.

Post-mortem examination.—The cardiac region of the stomach was greatly congested, the mucous membrane being spotted all over with blood. The mucous membrane of the intestines was intensely red. The gastric tubes were very much distended with granular matters and small nucleated cells about $\frac{1}{4000}$ part of an inch in diameter, but no normal gastric cells could be discovered. Between some of the tubes there were also elongated nuclei and a few small cells. The mucous membrane of the duodenum and ileum was everywhere loaded with small cells and granular matters. The villi were very erect, exceedingly large, and deeply coloured by blood, which seemed to have been extravasated

into their texture, especially at their free ends. They were loaded with small cells and granular matters. The tubes of Lieberkühn were distended with cells and granules. The pancreas was loaded with small cells and granular matters.

Although in all the above cases there was redness of the stomach after death, the amount of disease actually existing in the mucous membrane could not have been suspected had it not been for the microscopic examination. In the next cases, however, the morbid appearances were sufficient to show the nature of the complaint, without the necessity of a more minute investigation.

Observation 8.—In 'Beale's Archives' a case of scarlatina is reported, in which the patient vomited up with some bile "a piece of the epithelial coat of the stomach, about the size of the palm of the hand." At the post-mortem examination "the mucous membrane of the stomach and duodenum was much congested, but there was no extravasation of blood in any part. The muscular coat towards the pyloric orifice was firmly contracted, and the mucous membrane thrown into rugæ, but the cardiac portion was relaxed and the coats in this region seemed very thin."

Observation 9.—The stomach of a child who had died of scarlatina was kindly sent to me by Dr. B. W. Richardson. The stomach and duodenum were intensely inflamed; indeed, excepting the greater brightness of the colour, the mucous membrane presented more the appearance of a case of catarrh arising from heart disease than that of scarlatina. From the fact of the stomach having been preserved in ammonia, I was unable to make any microscopic examination.

Observation 10.—The small intestine of a child who died of scarlatina is preserved in the museum of the Middlesex Hospital, and there are numerous ulcerations which seem to have arisen in the solitary glands.

In all the above cases death took place within a few days after the attack of the fever, and, in the first, vomiting and diarrhœa preceded the rash. In Dr. Beale's case the vomiting and sore throat are mentioned as occurring on the same day as the rash, but I have generally observed symptoms of gastric disorder before the soreness of the throat has been complained of. It is, therefore, probable that the inflammation of the digestive tube takes place before or at the same time as that of the throat, and is, therefore, prior to the eruption.

The next cases not only strengthen the general conclusion as to the existence of inflammation of the stomach and intestines in scarlatina, but also show the changes which the mucous membrane undergoes in the later stages of the illness.

Observation 11.—A female child, æt. 9, was admitted into the Middlesex Hospital. Two or three weeks previously she had been affected with scarlatina. She died of diseased kidneys, with fluid in the chest. The mucous membrane of the stomach was not injected, but was covered by a thick gelatinous mucus. The tubes were easily separated from each other, and were not thickened. At their closed ends the gastric cells were obscured by a large amount of granular matters, but towards their free extremities the cells were plainly seen, about $\frac{1}{1200}$ of an inch in diameter, round and nucleated, but very much fewer in number than in the natural state.

Observation 12.—A child two years of age passed through the first stage of scarlatina, but died a fortnight afterwards.

Post-mortem examination.—The mucous membrane of the stomach was of a pale colour, and seemed thin and wasted, but in no part did it present any appearance of post-mortem solution. The tubes of the stomach were smaller than usual, and did not show the distended appearance observed in many other cases when death took place

earlier in the disease. They were lined by a membrane, but many normal cells were visible. After long maceration in spirit the tubes assumed a more natural appearance, and a larger number of cells became visible, although their outlines remained indistinct. In some sections the blood-vessels situated between the tubes were evidently enlarged, but this was by no means general.

Observation 13.—A man twenty years of age was attacked by scarlatina. He was admitted into the Middlesex Hospital, suffering from dropsy, and died about three weeks after the commencement of the fever.

Post-mortem examination.—The stomach was covered by a thick gelatinous mucus. It was not much congested, although the blood-vessels seemed large. At the commencement of the pyloric region the mucous membrane was thick, red, and softer than elsewhere. The tubes seemed loaded with granular matter, but also contained cells, although there were much fewer than usual. There was less than the usual adhesion between the tubes and their contents, for the cutting of the tubes obliquely generally caused them to appear empty.

Observation 14.—A boy was admitted into the Middlesex Hospital. When first attacked, he had suffered from repeated vomitings, but neither sore throat nor eruption on the skin had been observed. He died in two or three weeks after the commencement of his illness.

Post-mortem examination.—The back of the tongue and arches of the palate were stripped of epithelium, but there was not desquamation of the cuticle. In some parts the tubes of the stomach were greatly distended with granular matters, in others the cells were apparent, but seemed matted together and their edges obscured. In other places the tubes themselves were united together, and the spaces below them were loaded with nuclei and small cells about $\frac{1}{3000}$ of an inch in diameter.

Observation 15.—A man was admitted into the Middlesex Hospital, suffering from scarlatina, which was followed by peritonitis, bloody and albuminous urine, and constant vomiting. Death took place about three weeks after the invasion of the disease.

Post-mortem examination.—The stomach was covered by a thick tenacious mucus, and the mucous membrane was in some parts very dark in colour, almost black; in other parts red and greatly congested, and looked as if blood had been sprinkled upon it; in the middle region it was pale. The tubes were easily separated from each other, and greatly distended by granular and fatty matters. At the closed ends of the tubes the cells were scarcely visible, but were more readily seen when examined towards their free ends. The cells seemed cemented together and covered with fatty granules, and their edges were very indistinct.[1]

The first effects of the scarlatina poison upon the stomach seem to be to congest the blood-vessels, and to strip the epithelium from the tubes and the surface of the organ, at the same time that the tissues are softened. The tubes are greatly distended by granular and fatty matters, or by cells intermixed with granules, whilst in other cases they are lined by a newly formed membrane. Sometimes no normal cells can be distinguished; in other cases they are present, but are scattered irregularly. After the second or third week the tubes are found less distended, their closed ends are still loaded with granular matters, which greatly obscure the gastric cells, but these become more evident towards

[1] The following case has lately come under my notice. A girl, æt. 17, was attacked by scarlatina, and died upon the tenth day. The tubes of the stomach were very much distended by granular matter, and also contained cells which were larger and more granular than natural.

In about two feet of the small intestines, which I had the opportunity of examining, the mucous membrane was covered with a thick tenacious mucus, and only fragments of villi could be discovered, the greater part being quite bare of them. Under the microscope it presented the appearance of a sieve, being everywhere perforated by the enlarged and thickened openings of Lieberkühn's follicles. (See Plate VI, fig. 1.)

the free surface of the mucous membrane. The cells at this period are sometimes very large, sometimes loaded with fat or coated with granules, and seem still to have but little adhesion to the basement membrane, as they readily separate from the tubes, but adhere closely to each other.

The effects of inflammation upon the intestine seem, in slighter cases, to consist in the effusion of granular and fatty matters into the mucous membrane, but in more severe cases the tubes of Lieberkühn are choked with epithelial cells, whilst extravasation of blood takes place in the villi; and these, with the rest of the mucous membrane, are loaded with small cells and granules. In one case the villi were completely destroyed, and the tubes of Lieberkühn were greatly enlarged and distended by cells and granular matter. In some instances in which the pancreas has been examined appearances of inflammatory action have been remarked.

Tubular inflammation of the stomach is, however, a common disease; and the suspicion may be naturally excited that the appearances above described are nothing more than what might be observed in any other acute disorder.

Out of 150 cases in which I have carefully examined the stomach with the microscope, I have not remarked this condition to be so invariably connected with any other complaint, whilst the coexistence of inflammation of the œsophagus, stomach, and intestines, with a similar affection of the throat and skin, clearly points to a general cause.

In addition to this, the appearances presented by the stomach-tubes in scarlatina, when examined in the first stage, often differ considerably from those of ordinary tubular gastritis. The tubes are much more enlarged, especially at their closed ends, and are evenly distended, instead of presenting an uneven outline.[1] The basement membrane is not so generally thickened, and, in place of being cemented together into little lumps, the gastric cells

[1] It is well to state, perhaps, that the most characteristic appearances are met with in young subjects, who have succumbed in the earliest stages of the fever.

have either disappeared or are quite obscured by newly formed membrane or by fatty and granular matters. The differences between this form and gastritis as it ordinarily occurs, may be observed by comparing Plate V with Plate VI, fig. 2, which represents a section of the stomach taken from a child who died of convulsions on the third day after being attacked by measles. In this figure the normal gastric cells are plainly visible in the tubes, whilst between and below the tubes are numerous nuclei resulting from the inflammation.

As the above sixteen are all the post-mortem examinations I have been able to obtain of persons who have died of scarlatina; and as in all of them the mucous membrane of the digestive tube was more or less inflamed, I think it probable that this condition is a general accompaniment of the fever. As, however, the inflammation varied greatly in degree in different persons, and also in different organs in the same individual, it will, I think, be found that the intensity with which the mucous membranes are attacked varies according to the type of the epidemic and the age and constitutional peculiarities of the person suffering from the disease. The severity of the affection of the stomach and intestines is not necessarily in proportion to that of the skin and throat, for in some of the above cases little redness was observed in the latter when the former were intensely inflamed.

II.—*That desquamation of the epithelium of the stomach and intestines takes place in scarlatina.*

It is more difficult to prove that after scarlatina desquamation takes place in the digestive tubes as it does on the skin, for in the majority of cases vomiting only occurs at the outset of the disorder, and I have not had the opportunity of examining the matters ejected from the stomach in this stage of the disease. In Case No. 15 I found casts of the tubes in the fluid vomited, and from this I anticipated the appearances presented on post-mortem examination.

The chief reason upon which I ground the opinion that desquamation of the epithelium is of common occurrence is derived from the microscopic examination of the contents of the stomachs of those who have died of the fever, and the following are examples of the forms of casts of the gastric tubes as they appear under these circumstances.

In Case 1 there were a few ounces of a brown-coloured turbid fluid in the stomach, which had an alkaline reaction. The thicker portion of the contents, when placed under the microscope, was found to consist of pieces of fine membrane, of cells, granules, and shreds of membrane. The pieces of membrane were everywhere dotted over with granules, some of which were of fat. They varied very much in size, some of them were $\frac{1}{500}$ part of an inch by $\frac{1}{850}$ of an inch, and in many cases a few cells were attached to their surfaces. The cells, of which the contents mainly consisted, were very similar to those found in a healthy stomach. They varied in size from $\frac{1}{1200}$ to $\frac{1}{2200}$ of an inch, but were more generally about $\frac{1}{1500}$ of an inch in diameter; they were oval or circular in form, and flattened at the sides; they generally contained a nucleus and numerous granules, and were usually attached to fine shreds of membrane. After the addition of acetic acid the membrane became very transparent. (See Plate VII, fig. 1.)

In Case 12 the stomach contained a small quantity of turbid fluid, like gruel. When examined by the microscope it presented numerous pieces of membrane covered with cells, and separate cells, with ragged pieces of membrane hanging to them. The pieces of membrane were of different kinds; some thin, and presenting no appearance of structure, but covered with granules, and with a few nuclei adhering to them, whilst others seemed to consist wholly of cells bound together. In some cases it appeared as if the cells had been stripped from the tubes in a mass, the upper, wider, and flatter part being covered with conical, and the lower, longer, narrower, and more tubular portion being composed of round or oval, cells. The length of these casts varied greatly, and the width was from $\frac{1}{500}$ to $\frac{1}{1000}$ part of an

inch; but when their folds were flattened out by the compressor they were much broader. The free cells were like those of a healthy stomach, and varied in size from $\frac{1}{1000}$ to $\frac{1}{3000}$ part of an inch, but usually were $\frac{1}{1800}$ of an inch in diameter. Very few of them had a clear edge, but seemed attached to ragged pieces of membrane, and thus two or three cells were often joined together. (See Plate VII, fig. 1.)

In Case 5 the contents were found to consist chiefly of masses of conical epithelium stripped from the mucous membrane in large flakes. The epithelial cells were overlaid with fine granular matter. There were also numerous casts of the tubes, composed of fine membrane.

But the question arises, whether we can trust to such appearances in an organ so subject as the stomach to post-mortem changes. In order to ascertain this, I examined microscopically the contents of the stomachs of forty-five bodies inspected at the Middlesex Hospital, marking in each the condition of the mucous membrane. In one instance only were there any fibrinous casts like those above described, and it occurred in a case of acute gastritis. In eighteen cases there were only separate cells, chiefly of the columnar form; or if there were casts there were very few, and in none of them was there any inflammatory action of the mucous membrane. In eight cases casts of the upper parts of the tubes were plentiful, composed only of healthy conical cells; and in all these the mucous membrane was in a natural condition. In eighteen there were either plugs formed of cells and granules from the secreting parts of the tubes, or the casts of conical cells were overlaid with granular matter, and the stomachs were more or less inflamed. But although I have only found casts of the tubes in one case of scarlatina during life, I have met with them in other diseases in which inflammation of the stomach was present, of which the following are examples.

Observation 16.—A man about 55 years of age had for some years suffered from symptoms attributed to disease

of the kidney. A fortnight before his death he was attacked with constant vomiting, together with a scalding pain at the epigastrium and between the shoulders, increased by every kind of food. The vomited matters contained fibrinous casts of the stomach-tubes, and latterly, blood.

Post-mortem examination.—Both kidneys were diseased (morbus Brightii), and the stomach was much congested and covered with a thick layer of mucus. When examined by the microscope the tubes were found loaded with granular matter, and their cells cemented together, and with difficulty distinguished.

Observation 17.—A man consulted me suffering from violent pain at the epigastrum after eating, and only relieved by vomiting. The matters vomited early in the morning contained torulæ and numerous casts of the gastric tubes, composed of mucus, covered by small cells. Some I measured were $\frac{1}{33}$ of an inch in length, and $\frac{1}{363}$ of an inch at their closed ends; but towards their free extremities they were only $\frac{1}{371}$ of an inch in diameter. I have also met with tube-casts in cases supposed to be bilious attacks, and in cases of persons suffering from inflammatory dyspepsia.

If, then, we can discover casts of the tubes during life in cases of gastritis, and if, in scarlatina, this condition exists, and casts have been found in the stomach after death, there is, I think, very great probability that desquamation of the epithelium does take place in this organ, as it does on the skin and the kidneys.

The appearances of the stomach-tubes in the foregoing cases of scarlatina corroborate this opinion. In all of them there was a deficiency of the ordinary gastric cells, which were replaced either by small cells or granular matter; and in many instances the contents of the tubes seemed to separate with unusual ease from the basement membrane.

III.—*That, notwithstanding the anatomical changes in the mucous membrane of the stomach, the formation of pepsine is not prevented.*

The following experiments prove that the characteristic secretion of the stomach is not destroyed by the injury inflicted upon it by scarlatina.

Two drachms of the mucous membrane were beaten up with one ounce of distilled water and filtered, after standing four hours in a cool place. To this acid was then added, and the albumen was digested in the mixture.

Ten grains of albumen were digested at a temperature of 90° for twelve hours in an infusion of the mucous membrane taken from Case 7, to which three per cent. of hydrochloric acid had been previously added. Seven and a half grains remained, but in a very pulpy state. A similar experiment was performed with mucous membrane from Case 6, and six and a half grains were left, but very soft and pulpy. Both of these cases had died within the first week of illness, but in a third, who died of congestion of the kidney in the third week, a similar experiment showed a loss of five grains. The average loss was, therefore, three and two third grains. Of eleven males who died of various diseases at the same hospital, similar experiments gave an average loss of four grains, so that little diminution in the amount of pepsine had been produced by the scarlatina. In contrast to this are the results of similar experiments upon four cases who died of typhoid fever. In two the albumen had gained three grains of weight by imbibition, and was not at all softened; in two others it was softened, and in one it had lost half a grain, in the other a grain and a half. But the activity of the digestion must depend, not only upon the relative amount of pepsine, but also upon the bulk of the mucous membrane. The average weight of the mucous membrane of the stomach of ten males, dying of various diseases, I found to be eighteen drachms; that of the two

recent cases of scarlatina was eighteen drachms and sixteen drachms;[1] whilst it only amounted to fifteen drachms in one who died in the third week. But the loss of substance in typhoid fever appears much greater, as, in three cases I weighed, the mucous membrane only weighed eleven drachms in each.

I am not disposed to attach much importance to the results of the foregoing experiments, as showing the actual amount of pepsine in each case, inasmuch as I had not the means of examining the stomachs under exactly similar conditions, as to the date after death and the nature of the contents; neither would I lay much stress upon the deficiency of pepsine in the cases of typhoid fever as a characteristic of that disease, since I am unacquainted with the state of the patients during life; but as in these three cases of scarlatina the mucous membrane digested an amount of albumen equal to that usually dissolved in persons dying from other diseases, and in most of whom the organ was healthy as to its structure, it is certain that the characteristic secretion of the stomachs had not been destroyed by the inflammatory action.

IV.—*That the condition of the skin is similar to the condition of the mucous membrane of the stomach in scarlatina.*

I have only examined the skin in three cases of scarlatina. In the first, death took place after a few days' illness, and the only morbid appearance in the cutis was an occasional slight extravasation of blood in the neighbourhood of the sudoriferous ducts. The rete mucosum was greatly thickened; and numerous round cells with large nuclei were everywhere visible, intermixed with the natural cells. The basement membrane of the sweat-glands was thickened, and the epithelial lining so much increased that in most cases it obstructed their channels. In some of the sweat-glands the

[1] This was in a boy aged seventeen years.

coils of which they were composed were loaded with coagulated blood, and were greatly and irregularly distended. (See Plate VII, fig. 2.)

In the other recent case the appearances were similar, excepting that the external layers of the cuticle were stained with blood in minute patches, and the sweat-ducts were also reddened; but there was no extravasation of blood, either in the glands or cutis. In some of the ducts the epithelium was detached from the basement membrane.

In the case of a man who died in the third week the sudoriferous tubes were still choked up, but in the glands the epithelium seemed in many places to be detached, the basement membrane being bare, or covered only by ragged particles. The cutis was in a natural condition.

It will be remembered that the morbid condition in these specimens is similar to what has been before described as existing in the mucous membrane of the stomach, viz., an increase in the epithelium, with but little affection of the other structures; but we know that in scarlet fever the functions of both stomach and skin are greatly impaired; and as it has been found that the amount of pepsine is not lessened, may we not attribute the anorexia and the diminution of perspiration rather to the obstructed condition of the glands of these organs than to a loss of secreting power in the cells of their epithelium?

I have, in accordance with custom, described the appearances of the skin and mucous membranes as the result of inflammation; but the following considerations suggest the idea that the term, when so used, is perhaps misapplied.

We find in scarlatina that in each part the morbid condition is mostly confined, in the first instance, to the basement membrane, and consists in the formation of layers of new cells, which in the skin are transformed into cuticle of natural appearance, and in the stomach contain pepsine.

If future researches should prove that a similar condition occurs in the kidney and other organs, we shall have to look upon the structural changes produced as resulting from

increased physiological action, rather than from inflammatory action, and that the primary effect of the scarlatina poison is suddenly and violently to stimulate the natural cell growths of the various secreting organs; for though it is true that in scarlatina various internal parts are affected by inflammatory action, daily experience shows us that a sudden increase of the normal functions of an organ is apt to produce structural disease of the organ itself.

I would, in conclusion, beg to express my best thanks to Drs. Stewart, Goodfellow, and Thompson, the physicians of the Middlesex Hospital, for their liberality in permitting me to make use of their cases for the illustration of this paper.

DESCRIPTION OF PLATES V, VI, AND VII.

PLATE V.

The mucous membrane of the stomach in scarlatina.

a. The tubes, greatly distended.
b. A few cells still remaining.
c. Intertubular spaces, free from nuclei or cells.
d. Muscular coat.

PLATE VI.

Fig. 1.—View of the surface of the small intestine in a case of scarlatina.
a. Openings of the follicles of Lieberkühn, much enlarged and thickened. No remains of villi in this specimen.

„ 2.—Mucous membrane of the stomach in measles.
a. Tubes, containing cells.
b. Gastric cells, cemented together.
c. Nuclei and cells in the spaces between the tubes.
d. Nuclei and cells below the tubes. The cells between and below the tubes are not sufficiently numerous in this figure.

PLATE VII.

„ 1.—Tube-casts and cells from the stomach in scarlatina.
a. Tube-casts.
b. Separate cells.

„ 2.—Hæmorrhage into a sweat-gland, in a recent case of scarlatina. The section made in a slanting direction.
a. Sections of coiled tube of which the gland is composed.
b. A tube greatly distended and altered in shape.
c. The opening of a tube. The opening irregular in shape.

Tuffen West del. G.H.F.

W. West imp.

Plate VI.

Fig. 1.

Med. Chir. Trans. Vol. XLVII.

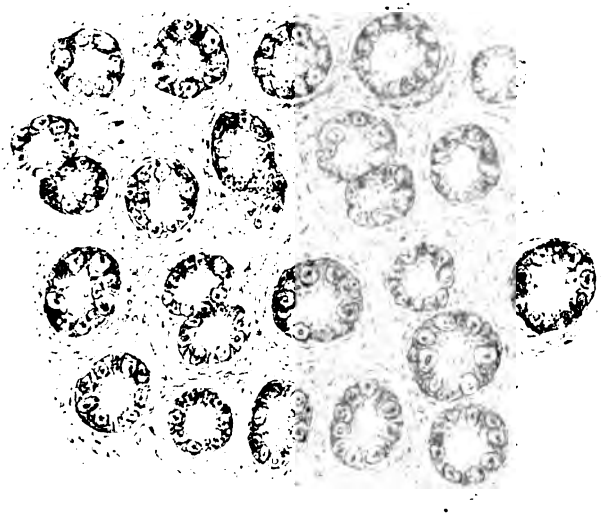


Fig. 2.

Tuffen West del. G H F

W. West imp.

Fig 1.

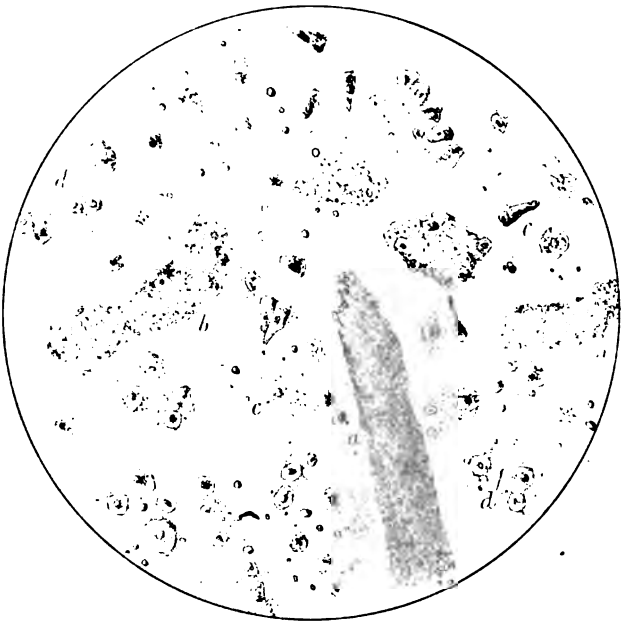
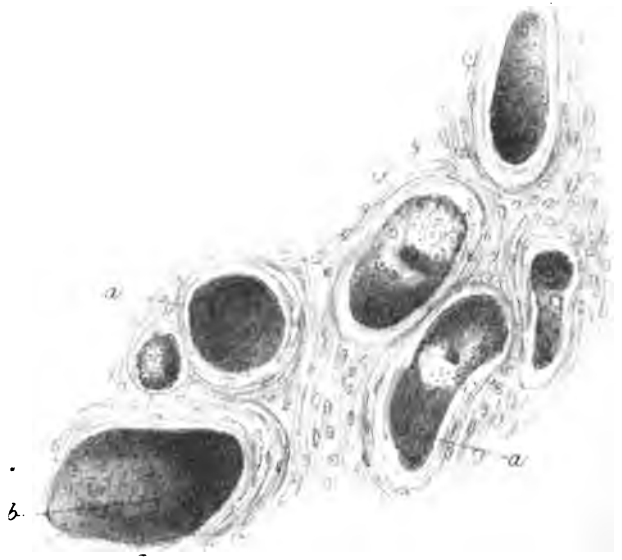


Fig 2.



Tuffen West. del.

W. West. imp.

ON THE

ORIGIN, STRUCTURE, AND MODE OF DEVELOPMENT

OF THE

CYSTIC TUMOURS OF THE OVARY.

BY

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Notwithstanding the increased attention which has of late been bestowed upon diseases of the ovary in consequence of the success which has attended the improved operative procedures adopted in this country for the relief of the cystic tumours of this organ, the pathology of these affections has still remained involved in considerable obscurity.

Believing, however, that an accurate knowledge of pathological anatomy will at all times contribute to the true advance of medical science, and feeling especially desirous, as a teacher in this department of knowledge, to be able to impart clear views on this important class of diseases, I have been led to an investigation of their nature, the results of which I now venture to lay before the Society.

For the means and opportunities of conducting these researches I am almost entirely indebted to the extreme

courtesy and kindness of Mr. Spencer Wells, who has most cordially responded to a request for assistance in procuring specimens, by liberally furnishing me with numerous instances of this class of tumours removed by him by operation, and has aided my investigations by every means in his power.

I have also to thank several other gentlemen for assistance most kindly furnished me in the investigation of the fœtal conditions of the ovary, and I have especially to mention the names of Dr. Graily Hewitt, Dr. Priestley, Dr. Randall, Mr. Squire, Dr. May, of Tottenham, Mr. Bennett, of St. Giles, and Mr. Roberts, the late clinical assistant in the midwifery department of University College Hospital.

I shall, I believe, avoid some confusion in the account which I have to give of my own researches, by first alluding briefly to some of the leading opinions which have been hitherto entertained with regard to the origin of ovarian cysts, more especially as I believe that some of them are capable of explanation by the phenomena which I have observed.

There is, however, one difficulty which occurs at the outset of an attempt to classify the opinions of the different authorities on this subject, and which arises in great measure from the want of a uniform nomenclature, and from the variety of terms in which diseases of the ovary, all characterised by the production of cysts, have been described by various writers. Since Dr. Hodgkin's[1] admirable description of these tumours, all those forms of cysts which do not contain secondary cysts or other growths in their interior, have usually been termed "simple," whether the cysts thus found be one or many, "simple" or "multiple" (Farre); but in the description of the more complex forms a great variety of terms have been employed—as "multilocular" (Farre); "cystoid" (Virchow); "compound cystoid" (Müller); "proliferous" (Paget); "composite" (Graily

[1] 'Med.-Chir. Trans.,' vols. xv, xvi; "Lectures on the Morbid Anatomy of the Mucous and Serous Membranes."

Hewitt). Kiwisch,[1] again, who has given a further classification of these formations, in terms which imply varieties in their mode of origin, and which have been pretty closely followed by Dr. Graily Hewitt[2] and Dr. Wilks,[3] distinguishes them as—(1) simple, (2) multiple, (3) multilocular, cystoid, or compound,[4] (4) alveolar disease of the ovary, (5) cysto-sarcoma of the ovary, (6) cysto-carcinoma of the ovary; and to these Mr. Spencer Wells[5] has added that of another variety, as "fibro-epithelioma, or alveolar adenoid tumour."

The views hitherto expressed by pathologists with regard to the origin of these cysts may be classified under the following heads:

I. That which attributes *all* cysts found in the ovary to a morbid alteration of the Graafian vesicle or corpus luteum, a view which has received the support of Carswell,[6] Andral,[7] Lebert[8] (who, however, is in some doubt whether a secondary cyst formation does not occasionally take place), Dr. Seymour,[9] Cruveilhier,[10] Dr. Hughes Bennett[11] of Edinburgh, Negrier,[12] and of Dr. Arthur Farre,[13] who has also given the best possible summary of the arguments in favour of this view.

[1] 'Diseases of Ovaries,' by Clay, 1860.

[2] 'Diagnosis and Treatment of Diseases of Women,' 1863.

[3] 'Pathological Anatomy,' 1859.

[4] These terms, according to a note appended by Dr. Clay to his translation of Kiwisch's work (p. 182), appears to be convertible, Dr. Clay adopting Dr. Farre's limitation (art. "Uterus and its Appendages," 'Cyc. Anat. Phys.') of the term "multiple," to cases where many simple cysts are developed simultaneously; "multilocular," to those which contain other cysts or growths.

[5] 'Path. Soc. Trans.,' vol. xiv, p. 205.

[6] 'Illustrations of Elementary Forms of Disease,' *Analogous Tissues*.

[7] 'Path. Anat.,' ii, p. 207.

[8] 'Physiologie Pathologique,' ii, p. 65.

[9] 'Illustrations of the Principal Diseases of the Ovaria,' p. 45.

[10] 'Traité d'Anatomie Pathologique Générale,' viii, 1856, p. 395.

[11] 'Ed. Med. Surg. Journ.,' vol. lxv, p. 400.

[12] Quoted by Dr. Tilt, 'Lancet,' 1849.

[13] 'Cyc. Anat. Phys.', art. "Uterus and its Appendages."

II. That which attributes the origin of the *simple* cysts, whether *single* or *multiple*, to the Graafian vesicles or corpora lutea, but regards such an opinion as untenable for the more complex forms, for which other modes of origin must be sought. This view has been maintained by Rokitansky,[1] Frerichs,[2] Bruch,[3] Virchow,[4] Führer,[5] Förster,[6] Scanzoni,[7] Bright,[8] Wilks,[9] West,[10] Graily Hewitt,[11] and Paget.[12]

Some of these authors, though regarding the secondary cysts as of new formation, believe that they are produced from the wall of the Graafian vesicles, which thus stand in the relation of parent structures to the secondarily formed cysts; Dr. Hodgkin, Mr. Paget, and M. Lebert support this opinion.

By far the larger number of observers, however, ascribe both the primary and secondary cyst-formations of this latter class to a new development taking place independently of the Graafian vesicles in the stroma of the ovary. Of the nature of such a process the most conflicting accounts are given. Thus, Dr. Bright (l. c.) regarded these growths as being of a "malignant" nature. Some authorities, again, as Müller[13] and Dr.

[1] 'Path. Anat.,' 1855, i, 230; iii, 412, 425. 'Wochenblatt der Zeitschrift der Gesellsch. der Aerzte zu Wien,' 1855.

[2] 'Ueller Gallert und Colloid Geschwülste,' 1847, p. 50.

[3] "Zur Entwickelungs-Geschichte der Path. Cysten;" 'Zeit. Rat. Med.,' 1849, p. 131.

[4] 'Verhandlungen der Gesellschaft für Geburtshülfe in Berlin,' 1848, pp. 220, 224; also 'Die krankhaften Geschwülste,' 1863, pp. 259, 260.

[5] 'Deutsche Klinik,' 1852, pp. 200, 201.

[6] 'Path. Anat.,' ii, p. 266.

[7] 'Lehrbuch der Krankheiten der Weiblichen Sexual Organe,' Wien, 1859, p. 355.

[8] 'Guy's Hosp. Rep.,' iii, pp. 180, 182.

[9] L. c., p. 411.

[10] 'Lect. on Diseases of Women,' 1858, vol. ii, p. 70.

[11] 'Diagnosis and Treatment of Dis. of Women,' 1863, pp. 383 et seq.

[12] 'Surg. Path.,' ed. 1863, p. 411.

[13] 'Ueber den feinern Bau und die Formen der krankhaften Geschwülste,' p. 55.

Wilks (l. c.), either express themselves as unable to account for their origin, or give no further account of the process.

Many of the more definite theories which have been brought forward to account for these formations, appear to be hypothetical applications to the cysts of the ovaries of more general views entertained by their authors on the subject of cystic growths, but some of them are of sufficient importance to deserve especial notice.

(A) That which has perhaps attracted the most attention in later years has been the opinion which has attributed the formation of cysts to a development from single cells ('Independent and Primary Anatomical Elements,' Rokitansky), by a process consisting in the enlargement of the cell, which, receiving a fibrous investment externally, then remains either a hollow cavity, or has a secondary formation of cells in its interior, which give rise to colloid substances by their metamorphosis. This view, supported as it has been by some of the most illustrious observers and philosophers in pathological science, has obtained a dignity among the theories of pathology which renders any detailed criticism impossible within the limits of this paper. Propounded originally by Dr. Hodgkin[1] in relation to the ovary, it has further, in regard to these tumours, received the support of Mr. Paget[2] and Professor Scanzoni,[3] while by Mr. Simon[4] and Rokitansky,[5] it has been held to explain the cystic formations in the kidney, and to it also has been attributed by Frerichs (l. c.) and Rokitansky[6] the origin of cysts in the thyroid gland.

It must be enough for me in this place to indicate that in many of the organs in which it was held to explain the origin of cystiform structures of new formation, other and simpler modes of development from pre-existing structures have been discovered. Thus, of the kidney it may at least

[1] 'Med.-Chir. Trans.,' vol. xxvi.

[2] L. c.

[3] L. c., p. 360.

[4] 'Med.-Chir. Trans.,' xxx.

[5] 'Ueber der Kyste,' p. 3, "Zur Anatomie des Kropfes;' and 'Path. Anat.,' ed. 1855, part i.

be stated that, after the researches of Gairdner,[1] Johnson,[2] and Otto Beckmann[3] on the origin of cysts in this organ from the renal tubules, the number of cases in which we have to resort to the cell theory to explain their formation must be extremely small; while those in the thyroid have been fully elucidated by Dr. Billroth,[4] and referred by him to the same type of formation (viz., the constriction of portions of tubular structures, through a return to the same embryonic conditions of growth[5]) as that by which the primary structures of the gland were first formed; and though opinions may still be divided on the nature of the so-called hydated mole, those given by Gierse,[6] Dr. Graily Hewitt,[7] and Virchow,[8] appear more in accordance with the facts of the case than the view of the origin of these structures from individual cells, as propounded by Mettenheimer,[9] and accepted by Mr. Paget.[10]

I must confess that I have never met with any facts tending to support this theory of cyst formation; and with regard to the ovary, I shall have shortly to give an entirely different account of the origin of the greater part of the cysts found in it, both in the more complex forms, and also of the small cysts occurring in the secondary papillary and dendritic growths which spring from the interior of large cysts, and which latter, though at first sight appearing most strongly to bear out Rokitansky's views of this mode

[1] 'Ed. Med. Surg. Journ.,' 1853.

[2] 'Med.-Chir. Trans.,' vol. xxx.

[3] 'Virch. Arch.,' vol. ix.

[4] "Ueber Fœtales Drusengewebe in Schildrusen Geschwülsten;" 'Müller's Archiv,' 1856, p. 144.

[5] Remak, 'Untersuchungen ueber die Entwickelung der Wirbelthiere,' 1853, p. 122; Kolliker, 'Microscop. Anat.,' vol. ii, p. 332; 'Entwickelungs-geschichte,' 1861, p. 392.

[6] 'Verhandlungen der Gesellsch. für Geburtshülfe,' 1847, p. 126 et seq.

[7] 'Trans. Obst. Soc. Lond.,' vol. i.

[8] 'Die Krankhaften Geschwülste,' 1863, p. 410 et seq.

[9] 'Müller's Archiv,' 1850.

[10] 'Lect. Surg. Path.,' 1863, p. 420.

of origin of cysts in such structures,[1] are, I think, susceptible of a different explanation.

(B) Another view, which has received the support of Vogel[2], Henle,[3] Velpeau,[3] Bruch,[4] Gierse,[5] Wedl,[6] and in a doubtful manner of Rokitansky ('Path. Anat.,' i, 230), regards cysts as spaces formed in areolar tissue, either pre-existing or of new formation, which spaces become filled with fluid either from serous effusions or fibrinous exudations, and subsequently become encapsuled by denser layers of areolar tissue, while an endogenous formation of epithelium takes place on their inner wall. This method, according to Wedl (l. c.), prevails both in the papillary growths occurring in the interior of, and also in the septa between the cysts.

(C) Closely allied to the foregoing is that described by Rokitansky[7] as the most common method of origin of the more complex forms of multilocular tumours of the ovary, consisting, according to him, of the breaking up of the stroma into a trabeculated structure ("Fachwerk," or panel-work), composed of membranous bands, uniting at their angles, and enclosing spaces or loculi filled with colloid or gelatinous matter, and giving rise to the peculiar *alveolar* texture seen on section of some of these tumours; these spaces which communicate with one another being, according to Rokitansky, the source of the peculiar complex formations met with in these cystoid growths. He explains the increase in the number of cysts by the continuous growth of these alveoli, which may either take place within the wall of the parent cyst (which then covers the secondary growth, giving rise to dense masses), or free in the interior of other

[1] 'Ueber die Kyste,' and 'Path. Anat.,' 1853, part i.

[2] 'Path. Anat.,' transl. by Day, p. 240.

[3] Quoted by Rokitansky, 'Ueber die Kyste.'

[4] "Zur Entwickelungs-geschichte der Path. Cysten.;" 'Zeitsch. Rat. Med.,' 1849, p. 131.

[5] 'Verhand. der Gesellsch. für Geburtshülfe,' ii, p. 182.

[6] 'Pathological Histology,' 'Syd. Soc. Trans.,' pp. 84, 460.

[7] 'Path. Anat.,' i, p. 109, fig. 48; p. 234, fig. 92.

cysts, when the growth presents a vesicular, loculated, or porous appearance ('Path. Anat.,' i, p. 236, fig. 95).

Scanzoni's[1] description of the origin of some of these tumours agrees very closely with Rokitansky's. As far as I can judge, Kiwisch[2] believes the condition described by him as "alveolar degeneration of the ovary" to originate in a very similar process; he says (l. c.) "that it appears as a breaking up of the stroma of the ovaries into cellular cavities, closely aggregated together. . . . . The cellular spaces do not apparently proceed from the follicles. Besides, the number of cellular spaces is so considerable . . . that we cannot easily trace their origin to the degenerated follicles; on the contrary, in general degeneration of the ovaries, the follicles appear to be mostly destroyed, so that they cannot be found as such. . . A partial alveolar degeneration of the stroma not unfrequently occurs in the intermediate tissue in cystoid disease."

Dr. Wilks describes, in similar terms, "alveolar degeneration of the ovaries" (l. c., p. 413), but he does not give any further account of the mode of origin of this disease. With great respect for the illustrious authors who have expressed these opinions, I have been led by my own investigations to believe that their descriptions apply rather to the appearances observed on section than to the mode of origin and *nature* of the process by which the secondary cysts are formed, a process which, indeed, Rokitansky admits (p. 234, l. c.) to be very difficult to discover. The relation of my observations to Rokitansky's views will be more fully detailed hereafter.

(D) Another opinion, also somewhat allied to the foregoing, and of considerable importance, is that expressed by Professor Virchow,[3] and which has been supported and further expanded by Professor Förster.[4]

[1] L. c., p. 355 et seq.

[2] L. c., p. 189 et seq.

[3] 'Verhandl. der Gesellsch. für Geburtshülfe zu Berlin,' 1848; "Das Eierstocks Colloid."

[4] 'Path. Anat.,' ii, p. 267 et seq.

Both these authorities agree in attributing a different origin to the "colloid" or "cystoid" disease of the ovary to that in which the single or multiple cysts take their rise.

Virchow, being struck with the prismatic or columnar appearance assumed by the colloid matter in the interior of these cysts, the columns being bounded by lines of fattily degenerated cells, attributes their formation to an *alveolar* origin, the lines of fattily degenerated cells representing the walls of alveoli, the other structures of which have wasted in consequence of the gradually increasing pressure of the colloid matter (*vide* fig. 1, l. c.). He says that spaces lined by epithelial cells are continually appearing in the stroma or walls of the cyst, and that the cells lining them may be seen in all stages of "colloid" change; but he expresses his inability to account for their mode of origin (p. 201, l. c.). He further explains the more fluid contents of some of these cysts as due to the further softening of the colloid matter thus formed.

Professor Förster gives a further account *of the mode of origin* of this process, of which I venture here to give a short summary, as I shall have hereafter to allude specially to his views; and it will, I think, be productive of less confusion if I introduce them in this place. He says that the cysts proceed from cells and nuclei of new formation in the stroma of the ovary, and independently of the Graafian follicles, which latter are destroyed during the formation of the cysts. "The neoplasm begins with an hypertrophy of the stroma of the ovary and a new formation of connective tissue, accompanied with a growth of cells and masses of cells in an embryonic connective tissue; while the latter is developed into a mature connective tissue, the cells and masses of cells become enlarged, and undergo a colloid transformation, the outer layers of these become epithelial cells, and the compressed layers of the connective tissue of the stroma represent the fibrous wall of the small cysts."

These views, both of Professor Virchow and also of Professor Förster, are undoubtedly minute and accurate descrip-

tions of the appearances observed. It is only with regard to the mode of origin of these spaces and masses of cells that I have to differ from Professor Förster.

(E) Another mode of origin of cysts of the ovary has been described by Rokitansky[1] as a "cysto-sarcoma uterinum ovarii." In the paper quoted below he describes one case in which glands similar to the uterine glands were found in the stroma of the ovary, and associated with small cysts; but he does not describe this as a general appearance of ovarian cysts. This condition corresponds very closely with that which I have quoted from Mr. Wells as forming a variety of these tumours, and termed by him "alveolar adenoid tumour."

(F) Of cysto-carcinoma of the ovary I have seen but few examples. In one case (which, through the kindness of Mr. Spencer Wells, I had an opportunity of examining), removed from a young woman, there were several small cysts in the cancerous mass, and others appeared to be forming. In two of these I could trace the remains of a membrana granulosa, which pointed to their origin from the Graafian follicles. Into others, a little larger, villous projections of cancerous matter were taking place through the partially invaded wall of the follicle. I could find in these cysts no traces of ova.

There is, I believe, no occasion for me in the present day to point out the distinctions or to dwell on the confusion which formerly existed between "colloid disease of the ovary," as this multilocular cystic formation has sometimes been termed, and the so-called alveolar or colloid cancer. The two structures have nothing in common beyond a certain amount of resemblance between the gelatinous matter contained in the ovarian cysts and the colloid or gelatinous intercellular substance of the cancer, a material which, however, is common to a large class of pathological products.

[1] 'Path. Anat.,' ii, p. 423; 'Zeitsch. der Gesellsch. der Aerzte zu Wien,' 1860, No. 37, Sept. 10; "Ueber Uterusdrüsen Neubildung in Uterus- und Ovarial Sarcomen." (I am indebted for the perusal of this paper to the kindness of Mr. S. Wells.)

It will be seen from the foregoing short and necessarily very imperfect summary of the views hitherto entertained on the origin of these cysts, that they involve the application of the most varied theories of cyst formation to account for the appearances observed; and, further, that the processes described are, if not incompatible with, at least perfectly distinct from, one another, and in no respects referable to one common type. It is only in the hope of being able to show that a comparatively uniform process does obtain for all these cysts, and reconciles and explains the opinions held by other observers, that I have ventured to occupy so considerable a space with a criticism of their opinions.

Although it is not improbable that aberrations in the formation of the corpus luteum may occasionally give rise to cysts, and instances of this have come within my own observation—yet I do not think either that this is a frequent occurrence, or that cysts thus originating attain a large size, or give rise to secondary growths; and it may, I think, be regarded as an established fact that both the *simple* and the *multiple*[1] cysts of the ovary do, in the majority of cases, take their origin from the Graafian vesicles. The weight of evidence which I have already quoted would be almost of itself sufficient to establish such an opinion, which has been supported further by direct observations of Lebert[2] on mares, Seymour[3] and Frerichs[4] on birds, and, finally, with an absolute proof by Professor Rokitansky,[5] who found altered ova still within the dilated cysts. The Graafian vesicle is, to all intents and purposes, a cyst with peculiar contents.

[1] I use the term "multiple" here, as elsewhere, in the sense defined at the commencement of this paper.

[2] 'Anat. Path.,' i, 243.

[3] L. c., p. 45.

[4] L. c., p. 50.

[5] 'Wiener Wochenblatt,' 1855.

In the calf they often attain a considerable size in the natural condition, and in the human fœtus of the eighth or ninth month they may sometimes be found of the size of a pea; and in the walls of these follicles, which are considerably distended with the liquor folliculi, numerous other follicles, in a less developed state, may often be found. The discovery of the ovum within certain of these follicles, when thus distended to cysts, is not always a matter of certainty, as recent observers, particularly Professors Grohe[1] and Pflüger,[2] have shown that the ova frequently disappear under circumstances unattended at least with any other pathological conditions. I have lately had an opportunity of observing, in the case of a female dying in Dr. Reynolds's wards, aged forty-two, both ovaries filled with cysts; in the left there were two large ones, each the size of two fists, another the size of an orange, and from this a series, in a descending scale, to those which only measured 0·01 of an inch in diameter. They were all lined with a partially flattened hexagonal epithelium, with distinct nuclei, and, with the exception that the epithelium in the larger cysts was flatter than in the smaller, they perfectly resembled each other, while the correspondence of the smallest with the walls of small Graafian follicles was complete. The other ovary did not contain any cysts larger than a pea, but these had a similar structure. I could only find in one instance (though there were from twenty to thirty cysts in each visible to the naked eye, and many more which were disclosed on examination with the microscope) any trace of an ovum, and this was somewhat uncertain in its characters, but the age of the patient in which they occurred might well explain their disappearance, as it is very probable, except in cases of protracted menstruation, that ova which remain in the follicles after the climacteric period in the female has been attained

[1] "Ueber den Bau und das Wachsthum des menschlichen Eierstocks;" 'Virch. Arch.,' xxvi, p. 302.

[2] 'Ueber die Eierstöcke der Säugethiere und des Menschen,' 1863, p. 76.

do undergo a process of atrophy probably analogous to the fatty degeneration of the epithelium of the seminal tubes in the male.

It is very certain that a *multiple* cystic tumour of the ovary may arise in this manner, and, further, that the distinctions between these and the compound cystoid forms are at times only to be separated by the artificial distinctions dependent on the number of cystic cavities produced; for there seems no absolute difference to be found in the structure of the cysts, and but little in the nature of their contents, which latter vary greatly, even in the same tumour, between the firmer colloid and the more fluid mucous or serous fluids, with their various other modifications arising from hæmorrhage or fatty degeneration of the epithelial lining or of the walls of the cysts themselves. If, therefore, any mode or modes of formation of secondary cysts can be shown to exist giving rise to the varied types of structure found in the multilocular or compound forms, and if the origin of these can be referred to cysts differing in no respect from those which arise from distension of the Graafian follicles, the conclusion may, I think, be fairly drawn that all the cystic diseases of the ovary take their origin in these structures. But although no precise limit has been established between what may be considered as constituting a multiple cyst, arising from the simultaneous dilatation of many Graafian vesicles, and those compound forms which have been hitherto referred to other modes of formation, (though the highly marked forms of the latter are at once distinguishable, as also are cases in which secondary or tertiary cysts arise from the interior of primary or secondary cavities), there is one point at once deserving of remark, viz., that although some varieties may be traced, yet that they all present essentially the same structure, viz.—(1) a wall of fibrous or connective tissue, corresponding more or less according to its thickness with the chief characters of the stroma of the ovary; (2) an epithelial lining, which presents many variations (to which I shall further allude) in the size and character of its cells; and (3) contents

which have been already very fully described by other observers.[1]

Both the simple and compound cysts have an external wall of the expanded and more or less altered stroma of the ovary; and I shall now proceed to describe such cysts, or parts of cysts, as have this both for their outer and inner boundary, though they may have in other portions of their interior groups or masses of clustered and compound cysts collected at various parts of the cavity.

The *peritoneal* surface of ovarian tumours may be perfectly natural, or it may present a considerable variety of appearances, according to the action, more or less inflammatory, which had existed externally, leading to roughness or adhesions. I shall not dwell further upon this, beyond remarking that it has occasionally small villous growths upon the surface, and that it is sometimes roughened by the opening, externally, of small cysts contained in the stroma, and a growth of their contents free into the cavity of the peritoneum. Perforation of the external wall by large papillary growths, such as Rokitansky and Mr. Hutchinson[2] have described, have not come within my observation, though I have seen minor degress of this condition.

The *external wall* of the cysts varies greatly in thickness. It may, in large and much distended cysts, be as thin as one twelfth of an inch, but more generally it is about a quarter to half an inch in thickness, and in portions corresponding to bands and trabeculæ, which cross the interior, it may considerably exceed this measurement. On section it is very commonly found to be more or less separable into two layers—an outer one, very firm and resisting, not easily torn with needles, and which is composed of a dense layer

[1] In this account I shall limit myself to the descriptions of cysts with "serous," "colloid," or "mucous" contents. I have been unable, while making these investigations, to procure any specimens of the dermoid cysts of the ovary.

[2] 'Path. Soc. Trans.,' xiv, p. 198.

of broad fibres running longitudinally in a direction parallel to the circumference of the tumour; and an inner one, having more the characters of areolar tissue, being softer, more easily torn, more fleshy and vascular looking, and seen under the microscope to be composed of a finely areolated network of fibres, in which are a large number of elongated nuclei and fibre-cells. These are sometimes thickly placed, and so arranged as to form a continuous tissue, in which the enlargement of each cell fits into the depression between the elongated portions of adjacent ones. The number of these nuclei and fibre-cells varies considerably in different preparations, probably in a direct ratio to the more or less rapid growth of the tumour, and the extent to which they are found in the deeper layers of the stroma, as compared with the external, points to its growth taking place chiefly in the former portion, while the latter must be considered as the older and more fully formed. This conclusion is not without interest in relation to some other facts, which I shall have shortly to adduce. (*Vide* Plate VIII, figs. 1, A, B.) The thin-walled secondary cysts also contain these elongated nucleated cells in large numbers. (Fig. *c* represents some of these.) The stroma of the wall contains very numerous vessels. The veins are particularly large, and are seen in great numbers in and immediately under the peritoneal surface. The arteries appear to lie more deeply in the stroma; they are of large size, and the larger ones have very thick walls. One notable peculiarity which they possess is that they retain in many cases the same twisted, corkscrew-like appearance which is seen in their course in the stroma of the ovary.[1]

Deeply imbedded in the wall, or projecting more or less either on its inner or outer surface, are frequently seen small cysts in various stages of development, and other elongated crypt-like processes, which I shall shortly describe more in detail.

[1] Grohe, loc. cit., p. 278.

The *inner surface or lining membrane of the cyst* presents a great variety of appearances. Leaving out of consideration for the present all the changes produced by secondary cysts on this inner surface, we find that two structures chiefly demand attention:

I. The epithelial lining.
II. Alterations in the innermost layers of the wall.

I. *The epithelium* presents many varieties. It naturally covers the whole of the inner wall, and in the greater part of its extent it presents a single layer of flattened polygonal cells, approaching more or less the circular form, but altered by mutual apposition. (Plate VIII, fig. 2, *c*, represents a typical specimen of such an epithelial lining.) The cell-walls are distinct; in the interior of each is a nucleus and nucleolus, and the cells appear as if imbedded in a clear or finely granular intercellular substance. They separate, in most cases, without much difficulty, and then assume a rounded form by imbibition. In other cases they strip off as a uniform membrane. Plate VIII, figs. 2, *c* and *e*, represent similar cells from smaller cysts, and others are seen in Plate X, fig. 42, G. In other cases the epithelium has a much flatter character, and is hardly distinguishable from the elongated cells of the connective tissue beneath, the nuclei of which, on addition of acetic acid, may be seen to form an almost continuous series of layers from parts of a section a short distance below the surface, until the surface itself is reached. It might almost be thought that the lining membrane had been denuded of its epithelium, and that on a vertical section we were only looking at the upper layers of the connective tissue, but I am satisfied that this is not the case, first, because these epithelial cells may be slipped off in many cases in an elongated form, and when isolated may swell up by imbibition and present rounded forms. Figs. 2, B, *b*, *b*, represent these appearances.

In other places the epithelium assumes a stratified character, and forms several layers. These may all continue of

a polygonal or rounded form, but more commonly this is retained only by the deeper strata, while the superficial ones assume a columnar shape, thus repeating a condition similar to that seen in the mucous membranes of other portions of the genito-urinary tracts. (*Vide* fig. 2, A.) At *a* is seen an isolated cell from the same object with two nuclei.

On making vertical sections with a Valentins knife through the epithelium into the layers of connective tissue beneath, the nuclei of the inner portions of this latter structure are seen gradually to enlarge and to become rounder as the epithelial surface is approached. The cell-walls corresponding to these nuclei in the connective tissue cannot be clearly made out, but at the line where the cells of the epithelium can be distinctly traced and immediately below this, there seems every form of transition between the nuclei or cells of the connective tissue and those of the epithelium, thus giving rise strongly to the impression that the cells of the connective tissue pass by insensible gradations into those of the epithelial structures in a manner probably similar to what Dr. Lionel Beale has described in the structures of the corium and submucous tissue of the papillæ of the tongue.[1] I have never seen anything like a membrana limitans by any methods of research which I have employed between the epithelium and the stroma of these cysts. (Fig. 2, *a'*, represents the appearance here described.) According to the character of its epithelial lining, the inner surface of the cyst has a glistening or velvety appearance. The latter character increases in direct ratio to the amount of stratification of its epithelium. When the wall has lost this covering, but without undergoing further changes, it

[1] 'The Structure of the Simple Tissues,' 64, 65, 109. It would be beyond the scope and foreign to the purpose of this paper to enter into minute histological details. I therefore think it best to confine myself to the terms already in use in the description of minute structures, only pointing out in a cursory manner such facts as I have clearly observed, though I abstain purposely from entering into a full discussion of their bearings on minute anatomy.

has a shining, more or less translucent, and semi-cartilaginous look.

Different portions of the wall of the same cysts vary in the nature and arrangement of their epithelial lining. Thus, in one part of a large cavity the epithelium may be stratified and columnar, in another spheroidal, and consisting of one or more layers, and in another portion it may present the flattened character already described, and which is with some difficulty distinguished from those portions which are already destitute of this covering. Further changes in the appearance of the inner membrane are due to fatty degeneration of the epithelium. This change may take place *in situ*, giving to the lining membrane the appearance of a thick, perfectly opaque, *uniform* layer, of an ochrey tint of various shades, spread over portions of the surface. It then appears chiefly to affect the spheroidal or flattened forms. The stratified layers, especially when they have assumed the character of a columnar epithelium, are usually cast off and float in the fluid as soon as the fatty change has commenced in them.

There appears to me to be in many cases a continual regeneration of the epithelial cells from below, attended with a shedding of the superficial layers into the contents of the cysts, which then give rise to the variously coloured contents found in them. When the epithelium is not regenerated the most internal layers of connective tissue are left bare, and they may then become the subject of further changes, the commencement of which may possibly have been the cause of the failure in the reproduction of the epithelial structures.

II. *These alterations in the innermost layers of the wall* consist in a fatty degeneration of the cells of the connective tissue, leading to calcification, which takes place in stripes and patches, and is so closely analogous to similar changes occurring in the aorta and cardiac valves that any further description of them here is unnecessary. I know few anatomical structures, however, where this change can be so

exquisitely observed. The appearance produced in the inner wall by this process is that of a series of ochrey patches, which differ from those produced by analogous changes in the epithelium by being more striated and less uniform.

To avoid repetition I would remark here that precisely the same change (at least as regards the fatty degeneration of the cells of the connective tissue) takes place in the walls of the thin-walled and secondary cysts, and leads to their rupture, either into each other or into the cavity of the parent cysts. Sometimes a convincing proof of this is afforded by finding a collapsed and empty thin-walled bag, which has ruptured, and the walls of which present an almost uniform fatty degeneration, affecting the enlarged cells of the connective tissue, in which all traces of nuclei have disappeared. The depth to which either the atheromatous or the calcifying changes may take place is very various, but presents nothing worthy of special remark. (*Vide* Plate X, fig. 41, and description of this figure.) Besides these changes the lining membrane presents other peculiarities, owing to the rupture of cysts into the interior of the parent cyst, or to the fusion of several into a single cavity. The septa and walls of these (the remains of which can sometimes be distinctly seen) gradually waste or become blended with that of the common cavity by continuous stretching, until no traces of them remain, except in the form of bands traversing the wall in various directions, giving rise to lines of elevation, of various thickness, in its interior. In addition to this there is very commonly seen, on the interior, a network of fine lines interlacing with one another, and forming a delicately areolated tissue, often with ochrey lines crossing it, and looking as if a fine piece of muslin net had been flattened into and incorporated with the wall. These are caused by the dehiscence of microscopic cysts into the cavity of the parent structure, the boundary wall of which has probably lost a portion of its vitality and powers of expansion. I shall allude further to this subject when these cysts come to be described.

Still continuing the description of the larger parent cysts (whose external wall is the stroma of the ovary covered by peritoneum), I have now to describe certain growths which take place from their interior, and which have earned for them the title of proliferous cysts. These are—

III.—Papillary or cauliflower or dendritic growths.
IV.—Villous growths.
V.—Glandular growths.

III. *The Papillary growths* have been most admirably described by Rokitansky and Dr. Hodgkin; but, as my observations have led me to form different conclusions with regard to their minute structure, and their relations to cysts which occasionally form in connection with them, I think it best to give an account of what I have myself observed with regard to them. They do not appear to be of very frequent occurrence. Thus, out of fifteen large ovarian tumours which I have minutely examined in all their parts, I have only found them in two, and both of these were removed in one operation, from the same patient, by Mr. Spencer Wells. The number of cysts discoverable by the naked eye in the ovaries in which they occurred was comparatively limited. In one there were only four or five large cysts, none exceeding a large turnip, and eight or ten smaller ones, from a crab apple to a hazel nut in size. In the others there were two cysts, one large, and capable of holding the head of a newly born child; the other, which was no larger than a Tangerine orange, was completely filled by one of these growths; while a few others, hardly observable by the naked eye, were seen under the microscope to contain similar structures.

The simplest form in which these occur are seen in Plate VIII, fig. 4, *a*, *b*, and fig. 5, *a*, as small, elongated, club-shaped elevations, from the inner lining of the wall of the parent cyst. The mode in which such a growth commences is seen at Plate VIII, figs. 13 and 14 (fig. 14 being a more advanced stage of fig. 13). The epithelium, closely adjoining, may be polyhedral or columnar (more commonly

the former), but presenting an approximation to the latter form; instead, however, of forming a uniform layer, the cells appear tilted up, and grow at an angle to the inner surface of the cyst, assuming an increasingly elongated form, containing large round nuclei in the deeper layers, while in the most superficial the cells are columnar. The growth, therefore, evidently appears to be one from the superficial strata of the stroma of the cyst-wall, and the connection of the epithelium on the surface with the deeper layers which belong to the connective tissue bears a great resemblance to that described by Dr. Billroth[1] in the epithelium of the frog's tongue, though I have not been able to find here the elongated spindle-shaped processes which he has described as forming a connection between the two kinds of tissue in the latter situation. Each of these papillæ have a rounded extremity; they contain a large loop of vessels; their stroma, as seen in Plate VIII, fig. 11, *b*, is of a delicate, hyaline, finely striated, membranous character, interspersed with numerous elongated nuclei. (This figure was taken from a specimen from which the epithelium had become separated in manipulation.) Plate VIII, fig. 12, shows a structure similar to those in figs. 4 and 5, *a*, *b*, but more highly magnified, to show its epithelial covering. The cells are all either of the spheroidal or columnar variety, most commonly the latter. They may swell by imbibition, and assume very large sizes (*vide* fig. 2, *d*), and they have a considerable tendency to undergo early and rapid fatty degeneration. These growths tend to form large composite masses through a simple repetition of the process by which their original growth took place, ensuing from the sides or extremities of those already formed, while at the same time each papilla or growth, whether of the primary or secondary variety, tends to enlarge in all directions. Various modifications of this process are seen in figs. 5, *d*, *d*, *d*; figs. 6, *a*, *b*, *b*; and in fig. 7. At fig. 11, *b*, is the stroma of one of these growths denuded of its epithelium,

[1] 'Müller's Archiv,' 1858, p. 159.

and consisting of a delicately fibrous network, with a clear intercellular substance and large nuclei. Some elongated nuclei, arranged in lines, mark the course of the vessels.

The vessels are very large in these papillæ, so that the growths are exceedingly vascular. If, when in a recent state, a fine stream of water is made to play upon them, they present a fine villous appearance, from the small secondary growths with free ends arising from their surface. Simple growths, or more or less multiple ones, may cover large tracts of surface in the interior of the cysts, or their formation may be limited to particular parts or patches. The size they may attain is considerable. One which stood alone in the interior of a single cyst was, when fresh, of the size of a Tangerine orange. This consisted of nine or ten lobes, some of which reached almost to its base, which rested on the floor of the cyst, while others were more superficial. The base was almost as broad as the free end of the tumour, the surface of which was very irregular. There was no pedicle. Around the base of this mass were scattered a group of smaller ones, varying in size from that of a hemp-seed to that of a bean. These growths were all distinctly solid. The inner and central parts were fibrous, but the outer and most superficial were delicately hyaline, and contained a few scattered nuclei; though here and there traces of striation, with fine loops of vessels and rows of nuclei marking the growth of new vessels into them, gave evidence of their solid nature. Further, there was no line of division between the hyaline ends and the more fibrous portions; and though they could be flattened by pressure, no fluid escaped on this operation, nor could any trace of a cavity be seen in them. The extremities represent newly forming and immature conditions of fibrous or connective tissue, as it is here that fresh additions to the growth take place from the deeper layers, which latter, being older and more mature, have acquired a more fibrous character. An illustration of this is seen in Plate VIII, fig. 11. Both in the base and also in the more superficial parts of these growths small cysts were seen, but usually these were but little dis-

tended; and on the mode of formation of these cavities I now wish to offer some remarks.

As adjacent growths approach one another the spaces between them, covered by the same secreting epithelial surfaces as the papillary growths themselves (which, in fact, constitute the boundaries of these spaces), become narrowed, so that two epithelial surfaces may be in perfect apposition or more or less separated at different parts.

Numerous illustrations of this condition are seen at Plate VIII, figs. 4, *c*, *c*, *c*; figs. 5, *e*, *e*, *f*; figs. 6, *c*, *c*, *d*, *e*; figs. 8, *a*, *a*, *c*, *d*, *e*. Further, a series of these masses, growing occasionally at the outset from narrow pedicles, bend over, and overlie the epithelial lining of the wall of the parent cyst, and, meeting at their sides or extremities, give rise to long, narrow, crypt-like spaces, of irregular shape and form, which are bounded by the adjacent papillæ and by the wall of the parent cyst. This is seen at fig. 7, where *c* represents the wall of the parent cyst; *a*, *a*, the narrow pedicles whence two adjacent papillary growths have sprung, which, giving off numerous irregular secondary growths, and at the same time overlapping the wall of the parent cyst, have produced the irregular spaces *b*, *b*, *b*. As these growths press against one another they adhere where the apposition is most complete, and grow together, according to well-known physiological and pathological laws (for though this is not the case ordinarily with surfaces covered with epithelium in a natural condition, it frequently takes place when such surfaces are in a high degree of irritation or abnormal nutritive activity), and we then get spaces more or less completely enclosed, and lined by an epithelium similar to that of the parent cyst. Such are seen at *d*, fig. 6, and *a*, *a*, fig. 10; while at *a*, *b*, *c*, *d*, *e*, fig. 9, all stages of this process may be seen in one preparation.

Further, it must be borne in mind that the secretion which is continually taking place into the interior of the parent cyst exercises an expansive force, which is tending continually to widen the area of the bases upon which these growths rest, by stretching the wall of the cavity.

This force, which would expand the wall until it became of excessive tenuity, is compensated for by a continual growth of the inner layers of the stroma, which appears to extend upwards into the bases of these growths themselves; and thus the spaces formed in the manner I have described tend constantly, not only to become elongated, but also are more and more removed, both longitudinally and vertically, from the site of their original production. Fig. 8, *c*, *d*, *e*, gives various illustrations of this process, which occurred in one preparation. The continuity between the forming cyst and the parent cavity is seen to be still partially maintained both at *d* and *e*. Fig. 6, *e'*, shows one of these cysts completely separated from the structures in which it took its rise.

I have thus attained, I believe, to a complete explanation of the mode of secondary cyst formation as occurring in these papillary growths. Each of the secondary cysts thus formed appears capable of having processes repeated in it identical with those which took place in the parent cyst; and the rapidity and facility with which this may ensue depends, in part, I believe, upon the amount of pressure to which they are subjected within the wall by the fluid contained within the parent cavity. I noticed in this preparation, before I had arrived at any clear comprehension of the process by which they had been formed, that nearly all the smaller secondary cysts in the wall were of peculiarly flattened and elongated forms, even after they had begun to expand by the secretion of fluid. Many of them contained secondary growths similar to the foregoing, springing from all parts of their walls, so as almost entirely, in some cases, to fill their cavity. I could not find in them any diverticula or offshoots, such as I shall have hereafter to describe in other cyst formations.

This mode of formation is, so to speak, an accidental one. It is only associated with irregular growths of these masses; and, as it requires peculiar combinations of position and growth of the surfaces which are to form the walls of the cyst-cavities, the production of cysts by means of it is not large. I have stated that they were few in number in both of

the tumours that contained these growths, and their position, whether at the bases or at the ends of the papillary masses, appears also in some degree to be a matter of accident. On section of the larger papillary tumours formed in this way, comparatively large areolar spaces may be seen, under the microscope, opening into one another, in all directions, and lined by epithelium, but they do not appear to be completely closed, although having much of the appearance of the alveolar texture described by Professor Rokitansky. I I do not, however, consider these as complete cysts, though any of them might become so. Plate VIII, fig. 15, represents a section made through such a growth.[1]

These papillary growths undergo various changes, mostly of the nature of a fatty degeneration, which give to the cysts containing them a very peculiar character.

The fluid found in their interior was, in all cases, of the consistence and appearance of very thick pea soup, but wanting in the tenacity and mucous character of other specimens in which these formations do not occur. Under the microscope it was seen loaded with free fat-granules, oil-globules, and the remains of fattily degenerated epithelial cells. Granule-cells and granule-masses were also frequent. I could not find in it any crystals of cholestearine. On looking for the source of these products, I found that many of the larger masses were more or less completely degenerated, the cells still adhering in some as ragged masses of an ochrey colour, which could be easily stripped from the subjacent structures. These curdy masses, which in some places have a great resemblance to rotten cheese, are found to be the secondary growths, or papillæ, which have degenerated *en masse;* and every gradation can be traced between the simple change of the epithelium on the surface to the destruction of the whole of the growth. When the change has once set in it generally extends through the whole thickness of the papillary growth, into the lining

[1] The appearances here described correspond very closely with those met with by Billroth in a polypus removed from the nose. 'Ueber den Bau der Schleimpolypen,' 1855, pp. 9—12.

membrane of the parent cyst, which is variously mottled by spots and patches of an ochre colour, slightly elevated, and which can be stripped off in parts (though with some difficulty) from the stroma beneath. The depth to which the wall of the cyst is invaded varies considerably; but while that process is taking place a great thickening ensues on the peritoneal surface, giving rise to patches of cartilaginous hardness, both externally as well as in the deeper parts of the wall. The size of these spots on the inner and outer walls of the cyst corresponds to the area of the base of the growths which have been affected. In one of the cysts which I have described there were three such patches, having each a diameter of rather more than an inch.

In one the layer of degenerated stroma had separated, leaving a depressed and intensely red surface, which was covered in spots with a film of amorphous exudation matter. The tissue surrounding all these was also intensely congested with spots of capillary hæmorrhage, and there was considerable ash-gray pigmentation in the immediate vicinity of the affected areas. The process appears to have been in all cases a somewhat acute one, depending, in all probability, though I could not trace this, upon some obstruction to the circulation in the growth, which had given rise to an almost necrotic action, while an inflammatory process was set up in the adjacent tissues, one effect of which, at least, as seen in the thickening of the wall beneath, was of a salutary kind, in preventing its perforation and the escape of the contents of the cyst into the peritoneum.

IV and V.—*Villous and Glandular Growths.*[1]

These processes are so closely allied, and, though distinct from one another in some respects, are of such frequent, I might almost say constant, occurrence, in conjunction with

[1] In the accounts of glandular formations I am compelled, for the reasons before stated, to omit all mention of those found in dermoid cysts, and which have been fully described by Steinlin, 'Zeitsch. Rat. Med.,' 1850.

one another, that I shall be obliged in a great measure to describe them together. In the glandular formations which occur in the interior of the parent, as well as in the secondary cysts, lies, I believe, in great measure, the clue to the explanation of the great variety met with in the multilocular or compound cystoid growths; though, in addition to these, I shall have yet to describe one other process by which this multiplication may take place.

The occurrence of villi in the lining membrane of ovarian cysts has been not unfrequently noted, and cases are given by Dr. Wilks,[1] by Luschka,[2] and by Friedreich,[3] where these have been covered with ciliated epithelium. This latter structure has never come under my notice in connection wtth these growths.

These villi commonly occur in patches of greater or less extent over the lining membrane of the cyst. They then give it, from their considerable vascularity, a delicately injected and at the same time a finely velvety character, which is very characteristic of their presence. They may occur scattered or in dense clusters; when in the latter condition, they are usually associated with glandular growths, in the formation of which their appearance constitutes the first stage.

When scattered, they do not commonly attain a large size, and appear to have little of the tendency to multiply by branching, which characterises the cauliflower growths last described. They rarely in such cases attain a greater length than from one twentieth to one sixtieth of an inch, and they differ markedly from the papillary and cauliflower growths in structure. While these last named are formed chiefly of a thick stroma of connective tissue, the villous growths contain but little of this, seeming, when denuded of their epithelium, to consist of little more than a loop of vessels, supported by a very small amount of connective tissue. Plate IX, fig. 16, represents such a growth of a few

[1] 'Path. Soc. Trans.,' vol. vii, p. 280.
[2] 'Virch. Archiv,' xi, p. 469.
[3] Id., xiii, p. 498.

scattered villi. They are covered more or less thickly with several layers of epithelium, which always seem to tend to assume the columnar form.

When more closely clustered, however, they lead to the formation of glandular structures of a nature allied to the crypts of Lieberkühn, the mucous glands of the stomach, or the glands of the uterus. I have carefully studied the mode of formation and growth of these glands, of which I shall now proceed to give an account. The first change which is noticeable in the immediate neighbourhood of a part of a cyst-wall in which a glandular formation is proceeding is, that a change takes place in its epithelial lining. Whereas in most parts this may consist of one layer of polygonal cells, it gradually becomes, as we approach the glandular growths, more stratified, and the superficial layer assumes a columnar character.[1] Into this stratified mass of cells delicate papillæ, with a fine loop of vessels, shoot from below; and, as these increase in length, a further growth of epithelium takes place at their free ends. We have thus a series of elevations, with corresponding depressions, formed in the epithelial lining, and these latter are converted into small tubular pits or hollows, at their bases, by a growth of the connective tissue of the stroma between the papillæ and around the enclosed masses of epithelium.

A series of tubular spaces is thus formed, the depth of which corresponds to the extent to which the stroma of the ovary has grown upwards. In its most superficial layers around these fossæ this latter structure is exceedingly delicate and transparent, so as to be seen with difficulty; but the hollow fossa at the base of the gland can be easily seen by separating the epithelial contents from the stroma, which can easily be effected after hardening the composition in chromic acid. The papillary loop of vessels, surrounded by a delicate stroma of connective tissue, rich in nuclei, can then also be distinguished, and the growth upwards of the stroma between the papillæ, and encircling the fossa,

[1] Somewhat similar appearances have been described by Steinlin, loc. cit., as occurring in dermoid cysts.

can be followed for a greater or less distance. (Plate IX, fig. 17, represents the first stage of the process; Plate IX, fig. 18, represents that last described.) As the growth of stroma proceeds upwards in the course of the glands, they become more and more completely enclosed, but at the extremities, when a further extension in length still seems possible, and when such an extension does actually take place, the projecting ends of the villi still retain their character as such. Plate IX, fig. 19, gives an accurate representation of two of a series of mixed villous and glandular growths, which occurred in a series of cyst formations, of which Plate IX, fig. 23, is a type, and which adjoined the one from which the figure now under consideration is taken. This figure is instructive also as showing a process by which these growths in some cases appear to be multiplied; for when these had acquired their tubular character, by the further growth from below, all the sinuses would represent diverticula from the gland. I have not, however, actually seen such a process completed.

We thus get a series of tubular glands with loops of vessels running in the delicate stroma between them, and which, as the glands become more deeply imbedded by the upward growth of the stroma, anastomose with other vessels which grow up in it (I believe, around their sides, or by means of other papillary loops), and form arches at various depths in the tissue. Such a structure is seen at fig. 20. These glands have an average diameter of $\frac{1}{100}$ to $\frac{1}{400}$ inch. They very commonly are lined by several layers of columnar epithelium, although occasionally they are found with only one layer in their interior. I have given this measurement from the centre of one papillary loop to that of another, as it is almost the only means of measuring the total diameter of the gland. The lumen, except under circumstances to be detailed hereafter, is very small, and not more than $\frac{1}{1500}$ or $\frac{1}{2000}$ of an inch. I find it therefore difficult to compare them with other glandular structures which are lined by only a single layer of epithelium, and of which the measurements are given by the various authorities on microscopic

anatomy. Their large size is, however, a very notable feature. I have never been able, either in the villous growths or glandular structures, to discover anything approaching the character of a membrana limitans. In recent specimens, and in those treated with Liq. Potassæ or acetic acid, no structure of the kind becomes visible; and in preparations hardened in chromic acid the epithelium may, as I have said, be stripped entire in a tubular form from the bases of the tubes without any such structure becoming apparent. I think it most probable that, in the more superficial parts of the tubes, the epithelium is supported, and the tube closed in the intervals between the papillæ, by some of the more delicate forms of connective tissue, but I have not been able to obtain distinct evidence of this with high magnifying powers (× 690 diam. of Powell and Lealand's microscope). I admit, however, that though I have carefully looked for it, I have not made it a subject of such minute and special investigation as to warrant a very positive and absolute expression of opinion on this point. Certainly, whatever the membrana limitans may be held to be, it is not seen with equal facility in these glands as in some others of the tubular kind.

The mode of formation of these glands differs, as far as I am aware, from that of any others, most of those of the skin or mucous membranes being described either as originating in the aggregation of rows of cells, or as formed from endogenous development in a single cell,[1] or as diverticula from the surface.[2] The peculiarity of those which I have described consists in their being primarily formed on the surface.

An increase in the number of these glandular tubes by lateral diverticula seems rare. One instance, however, of such a case is represented in fig. 24. As I said before, I have not seen such a process as is represented in fig. 19 completed. Another mode of multiplication is, however, very common; it is best seen in the glandular growths which

[1] Remak. [2] Kölliker.

fill the cysts of secondary formation, and which sometimes increase with exceeding rapidity. The process consists in an enlargement of the base of the gland, associated with the upward growth in its centre of a loop of vessels, forming, with a surrounding layer of stroma, a papilla similar to that with which formation of the primary glands first commenced. This may be continually repeated at the base of the same gland, which may thus be divided, inferiorly and for a certain portion of its length, by a series of septa, and so give rise to three or four divisions or tubes, all having a common outlet. This process may be well seen at Plate IX, fig. 21, *a*, *a*, and at figs. 23 *a* and 27 *a*.

Cystic formations from these glands may take place in several ways.

(*a*) The first and simplest is when the orifice of a gland, the base of which may be dilated and divided by septa in the way which I have just described, is compressed by the growth of glands in its neighbourhood, until the sides, being brought into close apposition, grow together, and finally occlude the opening. There is thus produced a closed cavity, terminated by a villous tuft which represents the corresponding sides of two adjacent glands, and which growth may extend considerably, and form one of a series of septa across the parent cyst, in which this formation has taken place. Such a mode of origin of a cyst is seen in Plate IX, figs. 21 *b* and 23, *b*, *b*; and the further growth of the septa is seen in Plate IX, fig. 23, *c*, *c*, *c*. It would appear that in some cases these septa themselves consisting of fibrous tissue carrying vessels, and covered with stratified epithelium, may thicken and give rise again to tertiary glandular and villous formations, which, in their turn, may go through the same series of changes.

(*b*) Another mode in which these glands may be converted into closed cavities is by their gradual dilatation into a series of very large crypts or follicles, across which, in a line more or less at right angles to the longitudinal direction of this cavity, septa may grow from their now thickened walls. An example of such a process is seen at fig. 22.

(*c*) We now come to the explanation of a process, in the elucidation of which I long experienced great difficulties, viz., the case where secondary cysts are enclosed, and spring from the wall of the parent cyst.

The formation of cysts of this variety is a somewhat complicated process, but one which I have so repeatedly and minutely observed, that I feel great confidence in the accuracy of the details which I now desire to lay before the Society.

Still keeping in view the wall of the parent cyst, it will be noticed that various sized groups of small cysts, single, or far more generally compound, project from their walls in numerous places. These may form very large and compound masses; but in a gradually descending ratio of size, some may be found which hardly exceed that of a millet or poppy seed, partially projecting from, and partially imbedded in the wall;[1] and on vertical sections other still smaller ones are frequently seen in great numbers with the microscope.

The cystoid character of these may be at once apparent to the naked eye, or they may present the appearance and feel of small solid masses, and even the microscope will sometimes only reveal a few small cavities in their interior. The causes of this latter condition will shortly be apparent.

Further, on careful observation, it will be found that these cysts nearly always exist in or near spots in the wall where the glandular structures which I have just described are also present. They are for the most part very rare and scattered in other places where the epithelium presents only a single layer, though their occurrence even in such spots may shortly be explained. Further, they are also rare when the villi only occur as scattered singly, and not in clustered growths united with glands.

Their origin in the places to which I have alluded appears to depend on the growth inwards of the stroma of the ovary (which originally caused the glands to become

[1] A good illustration of this condition is given in Rokitansky's work, 'Ueber die Kyste,' taf. i, fig. 1.

imbedded in the wall), proceeding to a further stage, and, finally, enclosing and shutting off the orifices of the glands themselves within its layers.

The commencement and extension of this process is seen at Plate IX, figs. 26 and 27. When the line of glands is single, as at Plate IX, fig. 26, the process is quickly accomplished, and either from the stroma itself, or by extension from the epithelium in the neighbourhood, a fresh growth takes place over the new surface thus formed. In parts where the glands and villi present a more complex arrangement, as in Plate IX, fig. 27, successive series of glands may be constricted off and encapsuled within the wall of the parent cyst, until we get a series of structures, as in Plate IX, fig. 28, corresponding to single glands and masses of glandular structures, the latter enclosed by thickened layers of the connective tissue and deeply imbedded in the stroma. The process seems partly aided by the previous conversion of some of the glands into cysts by occlusion of their orifices (in the manner described under *a*). These glands may have many divisions by septa at their base (so that the masses thus enclosed appear at first sight to have a very complicated structure, until the mode of their formation is clearly understood), and it appears as if the growth of the septa may continue, both in a longitudinal and transverse direction, across the cavities of the original tubes, *after* their orifices have thus been closed, and the glands grown over by the wall of the parent cyst, and thus *de facto* converted into cystic cavities. Such compound growths may be seen in Plate IX, fig. 28, *a*, *b*, *c*, *d*, and fig. 29, *a*. In some cases the origin of the cystic growth may still be traced by a long tubular process, either still open for a greater or less extent, or of which the previous course may be traced by a fibrous cord through the surrounding stroma. Plate X, figs. 30 and 31, give illustrations of this condition.

The glands and glandular masses thus shut off may be found in various stages of conversion into cystoid masses, protruding through or still imbedded within the stroma.

When a single simple gland has thus been closed and shut off, and the process of secretion continues, it forms a simple cavity; when a gland which has become more or less completely divided by septa undergoes the same process, a cavity with highly marked alveolar structures is at once formed. Plates IX and X, figs. 27, 28, 29, 31, and 32, give numerous illustrations of this process. The cavities thus formed have, of course, the same lining as the glands from which they spring, and from the moment of their dilatation into cysts they appear to have the same tendency to a stratification of their lining epithelium, and to the formation of glands in their interior, which marked the parent structure. Thus, Plate IX, fig. 29, is a more highly magnified representation of the cyst *e* in fig. 28, and thus the most complicated structures may be formed, the clue to which can only be found in the discovery, in the simpler forms, of the mode of origin of these growths.

Before discussing these I wish, however, to add a few words of explanation on the variations observed in the conditions of these glandular structures thus imbedded in the stroma. If a few and simple glands are shut off, we get on section round or oval masses of epithelial cells, with a wall scarcely distinguishable from the stroma by which they are surrounded, and in which they are deeply imbedded. (Such a formation is seen at fig. 32.) These may remain long quiescent, or, instead of developing into cysts, and retaining their lining membrane, the cells contained in them may undergo a fatty degeneration *en masse*, and nothing be found in the stroma but small round groups of fat-granules in which all traces of the original structure has disappeared. Such a condition is seen at Plate X, fig. 33. I do not know whether these may ever harden into cheesy masses. The most common course which they pursue appears to be, that their contents gradually become more and more fluid, and the walls undergo some analogous form of change by which they make their way to the surface, and rupture. A series of irregular alveolar openings are then produced, with small pits or depressions in the inner wall, opening into an

irregular series of cavities, the walls of which are destitute of any epithelial lining, and thus giving rise to a structure closely resembling Professor Rokitansky's figures and descriptions of trabeculated structures (Fachwerke), as the mode of origin of these cysts. It is with great respect to this illustrious pathologist that I venture to give this criticism and explanation of the appearances which he has described, and which would, if my deductions are correct, represent an aberration from and failure in the termination of cystic development, rather than the usual course which this process follows. Plate X, figs. 34 and 35, represent such alveolar processes as I have last described.

The cysts in the wall of the parent cyst may rupture at a very early stage, either internally or externally. The latter form of rupture on the peritoneal surface is, as far as I have observed, rare. When it takes place, the villous and glandular growths in the interior of the cyst protrude into the cavity of the peritoneum, forming a slightly roughened surface, attended with small pits and depressions. The villous growths do not appear, however, susceptible of any further growth into the cavity of the peritoneum. They soon lose their epithelial covering, and appear as short villous fringes of connective tissue, thus resembling those which may grow from the surface of other serous membranes. Whether or not they may give rise to adhesions with other organs is a question on which I am unable to offer any information. When, on the other hand, they rupture on the inner surface of the parent cyst, the villi and glandular growths in their interior do not appear, in all cases, to have such a transient existence, but may continue to grow into the interior of the parent cyst; usually, however, this process does not proceed to any great extent. The rupture of the wall appears to be associated with a condition of fatty degeneration of its fibres, which extends more or less into the stroma of the ovary, so as to give rise to a series of little depressions, bounded by lines of an ochrey colour, and which give a polygonal marking to the part of the wall on which it occurs. The area of the spaces enclosed

by these lines rarely exceeds one-twentieth of an inch in diameter.[1]

I believe that the account which I have just given furnishes a correct explanation of the origin of the alveolar structures lined by epithelium, which have been described by Professor Virchow and by Professor Förster as the source of the compound cystic degenerations of the ovary. They correspond in all points to Professor Virchow's descriptions, and the only point on which I have presumed to differ from my former illustrious teacher is, that whereas he describes the condition as a primary disease commencing in the stroma, I have endeavoured to show that it is a secondary condition of a kind to be considered more in detail hereafter, and commencing in the wall of the parent cyst. The explanation I have here given differs considerably from that of Professor Förster, though I think that the appearances I have described are identical with those to which he alludes. I have looked long and carefully for any indications of such a process as he describes, but in vain; in fact, it was at the outset of my investigations, and while searching the stroma for any facts confirmatory of his opinion, that I discovered the glandular structures in question, though it was long before I could arrive at a full understanding of the whole course and nature of these cystic formations. The process seems to be repeated almost indefinitely, even to cysts of the second and third formation, so that in the septa between the alveoli, and in all parts of the diseased structure, such cysts, and glandular growths giving origin to cysts, may in favorable specimens be found imbedded in the stroma. The best part to look for them is, however, in parts of the wall of the parent cysts, where there are only small nodules of the size of a millet seed protruding among villi and glandular growths.

In the secondary and thin-walled cysts the glandular and villous formations may proceed to an enormous extent, entirely filling their interior. Such a condition is seen commencing at fig. 23; but it may proceed to a much

[1] The appearances then produced correspond to a great extent with that figured by Dr. Bright, 'Guy's Hosp. Rep.,' vol. iii, part iv, fig. 2.

greater extent than is figured here. The containing wall may sometimes give way, and then we have apparently a free growth of glands, villi, and cysts mingled in great confusion; or, on the other hand, the cyst may continue to grow while its wall gains in thickness, and in such a case the glands in its interior may all or in part dilate to large follicles,[1] or crypts (of which fig. 22 presents an example), while the stroma between them also thickens, and we get a dense tissue perforated in all directions by alveoli, in which, occasionally, the intervening tissue may grow out of proportion to the glandular; so that, according to the predominance of one or the other of these processes of growth, we may get a cysto-sarcoma of the ovary, or the condition described by Mr. Spencer Wells as adenoma. A drawing of a section of such a mass is seen at Plate IX, figs. 24 and 25. This is apparently the condition which Professor Rokitansky has described as cysto-sarcoma adenoides uternum ovarii, (l. c.); but whereas he has described it as occurring in only one instance, I have traced it as the condition out of which secondary cysts and all the consequent varieties of structures in these diseases originate, in nine out of fifteen cases of ovarian tumours which I have examined. It is a noticeable peculiarity, to which I have already alluded, that isolated and sometimes comparatively small portions of the lining membrane of large parent cysts may become the seat of these growths and masses.

There is yet, however, another class of cases in which neither these glandular structures, nor, as a general rule, small nodular semi-solid cysts, can be found in the wall of the parent cyst, and in which, nevertheless, there is evidently a very large growth of secondary cysts proceeding. I have met with three such examples, and had great difficulties in accounting for their mode of growth, most of the secondary cysts being very thin-walled and transparent, presenting a great confusion of structure on section, opening

[1] Dr. Hodgkin, in his first paper on the subject, 'Med.-Chir. Trans.,' vol. xv, noticed the resemblance between "the opened cysts" and "*mucous* follicles on a large scale."

into one another in all directions, and greatly altered in shape by mutual pressure. Though not so densely crowded as those formed under the conditions last described, they still presented the alveolar appearance figured by Professor Rokitansky (in figs. 93, 94, pp. 234, 235, 'Path. Anat.,' vol. i). When, however, I placed a large tract of one of these thin-walled transparent cysts under the microscope, and viewed it with a low power, I at once arrived at an explanation of the manner in which the secondary cysts had originated.

Plate X, fig. 36, represents the appearances then observed. Projecting from the inner surface of the cyst, into and through its outer wall, there were seen in the same field three crypt-like processes, which were distinctly hollow, and lined by the same polygonal epithelium (ascertained by a higher magnifying power than that by which they are represented here) as covered the interior of the (to them) parent cyst. The opening by which they communicated with the cyst from which they sprang was very narrow, but they at once expanded into large flask-shaped sacs, which protruded into the cavity of the cyst adjoining the one from which they took their origin. It will be evident at once how indefinite a multiplication of secondary cysts may ensue by means of such a process. The complexity of these structures is further increased by adhesions between the outer walls of secondary cysts thus projecting into other cavities, and the inner walls of the latter with which they come into contact. In another and similar structure I found, however, in the dense stroma of the wall of the primary cyst (which was expanded into a large cavity, with only a single group of cysts at one extremity), another series of somewhat analogous processes. In the portion of the thick wall which adjoined the group of cysts just mentioned, I found the appearances represented in figs. 37, 38, 39, 40. These small cysts gave off sometimes one, sometimes more processes or diverticula, which after extending for a variable distance in the growing stroma, had a series of portions constricted off, so as to form shut cavities. I

believe that this process commenced at once in the altered Graafian follicles; and I find a confirmation of this view in the fact that in the case of ovarian-cystic disease, previously mentioned as having occurred under the care of Dr. Reynolds, and when I was able to trace every gradation between the Graafian follicles and the large cysts, the same process had also commenced. Plate VIII, fig. 3, gives an example of the origin of this process, taken from that case, where a series of such cavities are united by short processes. In the case also whence Plate X, fig. 36, was taken, I found a similar condition to that just described existing in the wall of the parent cyst. The process here followed, therefore, seems to be equivalent to the growth of glands by diverticula or budding, so common in all the glandular structures (including the lungs), which originate from the gastro-intestinal mucous membrane. I have endeavoured carefully to follow the growth of these latter structures, but only give my opinion upon this point with a certain degree of hesitation. At Plate X, fig. 39, the extension of the process given off from the small cyst, *a*, is seen at *c* to run into two parallel rows of cells, but these seemed gradually to pass into a single row, and to be lost in the layers of the stroma. In two instances, however, of one of which Plate X, fig. 40, is an example, I noticed the following facts. In the first place, I could nowhere discover any membrana limitans intervening between the epithelium and the stroma beneath; secondly, at the end where the extension and growth appeared to be taking place, the stroma did not present its usual appearance; in fact, it did not appear to form any boundary line to the epithelium at all; but in place of the fibrous tissue which surrounded the long crypt at all other parts, there was here seen, in the immediate neighbourhood of the epithelium (which was here almost columnar), a series of round nuclei, to which, however, I could discern no outer cell-wall; a little removed from this, there were brought out by acetic acid a group of oval nuclei, arranged for the most part with their long diameters converging to an imaginary line prolonged vertically through

the long axis of the crypt. Billroth[1] has seen a somewhat similar appearance in the connective tissue around the ends of ducts of new formation in a glandular tumour of the parotid; and his description, which I quote, corresponds very much to the appearances seen by me, although I could see no central fibre, as observed by him. "In these tumours peculiar cylindrical and flask-shaped structures are found, which often give the impression of a glandular substance, except that there is often seen in their centre a white line, which can be easily taken for a canal. These formations, however, proceed from the connective tissue, and the central white line is a bundle (or fibres? Bindegewebs-Strang), in which the spindle-shaped cells hang like the needles on a twig of fir. . . . The spindle-shaped cells, as I have lately absolutely convinced myself, may become converted into an elongated columnar epithelium, which begins to secrete, and so causes the formation of a cyst in the gland proceeding from the connective tissue." Such a statement, from so accomplished and accurate an observer as Dr. Billroth, would go far to explain the mode of extension of many glandular growths, especially those glandular hypertrophies which take place in the midst of old and preformed tissue; and the observations which I have just recorded would appear to be confirmatory of his view of the mode of growth of glandular structures of new formation. It is a subject, however, on which I have no further knowledge from direct personal observation than the facts which I have here recorded, and which does not afford to my own mind a convincing proof of the conversion of the stroma of the ovary into gland-tissue; though the observations which I made on the growth of the cauliflower masses of the villi and of the glands on the surface, as well as of the epithelial linings of some of these cysts, have led me strongly to incline to the conviction, which Billroth's observations on the papillæ of the tongue of the frog further confirm—that the epithelium may be merely a series of

[1] 'Beiträge,' p. 78.

metamorphosed and superficial cells of the so-called connective or fibrous tissue.[1] Still, however, without pressing this conclusion to such a length in the case now under consideration, it may at least be concluded, from the crowd of nuclei seen at the end of the tube, that a process of growth was taking place at this spot; and it may be considered possible that the rounder nuclei proceeded from the old, and were about to represent new epithelial cells, while those which were elongated represented a new formation of ovarian stroma, hereafter to form a fibrous wall for the glandular tissue.

The process has, however, a further parallel in that described by Langer[2] as occurring in the growth of the ducts of the mammary gland. He refers their extension to a conversion of the stroma of the mamma into glandular tissue. His figs. 9, 10, correspond very closely to the appearances which I have described as occurring in the growth of these crypts.

I may further mention that I have been able to ascertain that the two processes which I have just described, viz., the constriction of tubular cavities, and the increase of cysts by diverticula from the thin-walled specimens, have occurred simultaneously in the same specimens. The similarity of the processes is so great that this might have been predicted, and in the preparation whence Plate X, fig. 35, was taken, such processes as are represented by Plate X, figs. 37, 38, and 39, were found in the thickened wall of the parent cyst; and if it be objected that such an appearance as Plate X, fig. 39, represents could be presented by a cyst flattened by pressure, and seen in profile on section, there is at least no fallacy possible in the observation of diverticula such as are represented in figs. 37 and 38; nor in that in Plate VIII, fig. 3, where the stroma of the ovary

[1] I have retained this term as a provisional one, though aware of the exceptions taken to its use by many eminent anatomists, because at present we have none in common use by which it appears to me that it can be replaced.

[2] 'Denkschriften der Wiener Akademie,' vol. iii, 1852, p. 26.

in which they were found was subjected to no pressure whatever. In one of these cases, although I could discover no glandular structures forming in the inner wall, there were found, in a few isolated cysts apparently of older growth, scattered villi, which here and there tended to become clustered, and in several others the epithelium was stratified. It is easily conceivable that the consecutive stages of gland formation might have ensued here, and thus have greatly complicated the structure. This is also further of interest, as showing that *any* cyst, and even those isolated in the midst of more complex masses, may become the nidus for a multiple cyst formation; a point of great interest in relation to the questions raised between the relative value of tapping and total removal of these tumours.

The formation of secondary cysts by the constriction of portions of longer ones by bands of fibrous tissue is well illustrated in Plate X, fig. 42, where the preparation was taken from a secondary cyst formed in the wall of one of the tumours containing papillary growths, before described, and where across a constricted portion of the elongated cyst, *e*, a band of fibres, *f*, is seen crossing. The cyst *c* seems to have been already constricted off by a similar process (*vide* description of figures).

The only other changes which I have to notice as occurring in these cystic growths is their fatty degeneration *en masse*. This process, which often can be followed in minute cysts at the edge of the larger portions affected in this manner, gives rise to large tumours, from the size of a man's fist downwards, converted into hard, semi-solid masses of a fatty character, in which all traces of structure seem lost. It is more commonly met with in those where the contents were previously of a dense colloid-like nature than in those where they have had a more fluid character. The determining cause of this condition must lie, I believe, in some obstruction to the circulation, but I am unable to offer any direct proof of this presumption. I have also observed, in one instance, a very intense inflammation arise in the interior of a compound ovarian cyst consecutive to

tapping. It was attended with an extensive layer of exudation into and upon the lining membrane, which, when it took place, entirely destroyed all traces of glands and villi, in the parts where it occurred, though these were numerous in other situations. One notable feature about this inflammatory action was, that it had extended to other cysts adjacent to that in which I presume it had originated, and where the trochar could not by any possibility have penetrated. The appearances connected with such inflammatory action have, however, been so well described by Professor Simpson, of Edinburgh,[1] and by Kiwisch,[2] that it does not appear necessary to give any further account of them here.

On the chemical nature of the contents of these cysts I have little to add to the facts given by Professor Scherer,[3] Professor Virchow (l. c.), Dr. Owen Rees,[4] and by M. Becquerel.[5]

The amount of time consumed in the anatomical investigation prevented my undertaking any quantitative analysis of the contained fluids. I made, however, some observations on the reactions of the fluids contained, which I append for the sake of comparison with the results obtained by other observers.

On the fluid obtained from a thin-walled cyst the size of a walnut, which was one of a few projecting into the interior of a large, comparatively simple cystic cavity, I made the following observations:—

I.—1. The substance was comparatively fluid, being somewhat slimy; it was limpid, and, with the exception of a few opaque flocculi, transparent.

2. The reaction was faintly alkaline.

3. Acetic acid produced in the cold a slight clouding, not removed by excess of acid, but removed by boiling.

4. Boiling, after the addition of acetic acid, caused the

[1] 'Obstetric Works by Priestley and Storer,' i, p. 252.

[2] Loc. cit.

[3] 'Chemische und Microscopische Untersuchungen,' 1843. 'Verhand. der Phys. und Med. Gesell. zu Wurzburg,' vol. ii, 1852.

[4] 'Guy's Hosp. Rep.,' vol. iii.

[5] Clay's 'Kiwisch.'

fluid to become exceedingly frothy, but gave no precipitate.

5. Hydrochloric acid gave a very faint clouding; soluble in excess.

6. Solution of ferrocyanide of potassium added to the last named caused a copious precipitate.

7. A solution of bichloride of mercury caused no precipitate.

8. Neutral acetate of lead caused a very slight precipitate.

9. Basic acetate of lead caused a copious precipitate.

II. In a multilocular cystic tumour, examined November 30th, the fluids contained in the different cysts varied somewhat in character.

A. In a large cyst, with thin walls and clear contents, the fluid was very viscid, but clear.

1. Reaction very faintly alkaline; but, after standing for a short time, this reaction became more marked. (Qy. From evolution of ammonia?)

2. Sp. gr. 1010; 60° Fahr.

3. Acetic acid gave no precipitate in the cold.

4. Boiled with acetic acid, both when faintly acidulated, and with excess of acid, it gave no precipitate.

5. Hydrochloric acid. No precipitate.

6. Solution of ferrocyanide of potassium, added to last, gave a very faint clouding of an opalescent tint, which became darker on standing, thus indicating the presence of iron. (I have no note of the fact, but I believe that I tested the hydrochloric acid used at the time.)

7. Solution of bichloride of mercury gave no precipitate.

8. Neutral acetate of lead gave no precipitate.

9. Basic acetate of lead gave no precipitate.

B. In a second cyst having the same external characters, the contained matter presented considerable varieties. While one portion was comparatively fluid, refracting, and transparent, another part, though also clear and refracting, was very dense and semi-solid, approaching a consistence between that of the vitreous humour and crystalline

lens; and between the more fluid and solid portions every gradation could be observed. The more solid part dissolved after a time in Liq. Potassæ. The fluid portion was of a very pale straw-yellow, with only a very few flocculi of fattily degenerated epithelium within it. It was so excessively viscid that the sp. gr. could only be taken with difficulty.

1. Sp. gr., 1010 (?).

2. Reaction very faintly alkaline, with same characters as that last mentioned.

3. Boiling produced in it a striated clouding, not uniformly affecting the whole mass, nor redissolved by excess of acetic acid.

4. Acetic acid produced no precipitate.

5. Boiling with excess of acetic acid—no precipitate.

6. Boiling the fluid faintly acidulated with acetic acid caused a faint precipitate.

7. Hydrochloric acid caused a marked precipitate, soluble in excess.

8. In the solution thus acidulated with hydrochloric acid, ferrocyanide of potassium produced a marked precipitate.

9. Bichloride of mercury caused only the faintest clouding.

10. Acetate of lead (neutral) gave no precipitate.

11. Basic acetate of lead gave a marked precipitate.

III. In a thin fluid, examined December 10th, there were also great differences in the fluid contained in the different cysts. In some it was very slimy, and could be cut with scissors or a knife; in others it was perfectly fluid, and between these all varieties could be found. The average fluid contents were limpid, slightly opalescent, and containing but a few flocculi.

1. The reaction was strongly alkaline.

2. Sp. gr. 1015.

3. Boiling gave a marked precipitate without the addition of acetic acid.

4. Acetic acid gave no precipitate, either in the cold or on boiling.

5. Hydrochloric acid gave a precipitate soluble in excess.

6. Ferrocyanide of potassium gave a precipitate in solutions acidulated both with acetic and hydrochloric acids.
7. Bichloride of mercury gave no precipitate.
8. Sulphate of copper gave a precipitate soluble in excess.
9. Neutral acetate of lead gave a faint clouding.
10. Basic acetate of lead gave a moderate precipitate.
11. Solutions of alkaline salts gave no precipitate.

Imperfect as these results are, they yet tend to show that in these fluids there is a considerable difference between the contents of the different cysts. In all, the reactions obtained are more akin to those modifications of albumen discovered by Professor Scherer, and termed by him metalbumin and paralbumin, than to any of the hitherto isolated members of this series. The non-precipitation with acetic acid seems to distinguish them from mucus, though in the first mentioned a faint indication of its presence was thus obtained.[1] The absence of precipitates with bichloride of mercury is a notable fact, thus marking a difference between the reactions obtained by me and some of the reactions obtained by Professor Scherer.

It would seem as if very great varieties were possible in the condition of these fluids, and their varying reactions may probably be accounted for in a great extent by differences in the amount of pressure to which the secreting surface is subjected. The subject is one which appears to me to require further investigation, not only in relation to these cysts, but also with regard to those found in other portions of the body.

Professor Virchow[2] is of opinion that the more solid matter is always the first stage, and that dropsy of the ovary, or cysts filled with more fluid contents, represents a later stage of the same process, the more fluid matter being only the result of the gradual softening of the colloid material. In many of the younger and commencing cysts I have found the contained matters to be very firm; in

[1] '*Vide*' Hoppe. Anleitung zur Pathologisch. Chemisch. Analyse.'
[2] 'Eierstock's Colloid,' l. c., p. 213.

others, again, presenting similar characters, it was already much more fluid; and in the larger cysts, of the size of an orange, there is the greatest possible difference in the contents of adjacent cysts, of apparently the same age and structure with regard to the solid or fluid characters of the contained material. The impression which I have formed during these examinations is, that although Professor Virchow is perfectly correct in attributing some of the fluid contents to the softening of portions that were previously more dense and semisolid, yet that this process will not account for the whole of the more fluid contents of the larger cysts is an impression which I believe that Professor Virchow would also entertain.

Various alterations are found in the contained fluid, from the admixture of the fattily degenerated epithelium and walls of the cysts, as well from rupture of blood-vessels and hæmorrhage into their interior. These need no further description on my part, as they have been already well and fully described, and are familiar to every one in the habit of examining these structures.

Conclusions and Summary.

It remains for me to make a few remarks on the position assignable to these structures in a pathological classification.

I stated, in my remarks upon the opinions of those who held that all cysts of the ovary proceeded from the Graafian follicles, that this could only be proved by the discovery of the mode in which secondary cysts may arise from these structures. Of such methods of secondary formation I have, I think, given convincing proof;[1] and I would only again refer to the opinion which I have expressed before,

[1] Of all the *forms* of these multilocular tumours figured by Dr. Bright ('Guy's Hosp. Rep.,' vol. iii), Lebert, and Cruveilhier, I have seen instances associated with the modes of secondary cyst-development now described.

that there is no difference between the simple cysts of the ovary, which are admitted on all hands to originate in the Graafian vesicle, and the parent cysts of the more compound type, beyond those alterations in the walls of the latter which depend on the structures which give rise to the cysts of secondary formation.

This mode of secondary cyst-formation follows, with certain deviations, the now well-known type of the formation of cysts from the constrictions of portions of ducts in glandular structures, associated with the hypertrophy and fresh growth of tubular formations.

Of the mode of formation of glandular structures which I have described I can find no pre-existing instances in the authors on embryology or pathology whom I have consulted. Even in the mucous polypi described by Dr. Billroth[1] the formation of new glands appear to originate in diverticula from the surface. Such a process in the tense wall of a distended ovarian cyst would appear almost impossible, and this may possibly account for the difference in the mode of growth.[2]

When once formed, they have many parallels, not only in the whole course of the gastro-pulmonary and gastro-intestinal tract, but also more especially in the uterus, which must be regarded, from its embryonic development, as part of the natural duct of the ovary. This fact, as well as the occasional occurrence of ciliated epithelium in the contents of these cysts is, when we remember that the first part of this duct in the Fallopian tube is also provided with a similar epithelium, of the highest interest in connection with the views recently put forward by Dr. Pflüger, of Bonn (l. c.), on the embryonic condition of the ovary itself, in which he describes the origin of the Graafian

[1] 'Ueber den Bau der Schleimpolypen.'

[2] I do not fully understand the whole mode of formation which Steinlin (l. c.) has described as ensuing in the sebaceous glands of such cysts as contain dermic structures. His account of the formation of papillæ corresponds very closely to that in which I have here described the growth of villi.

vesicles in the calf and kitten as resulting from a series of constricted portions of the tubes or crypts of which the ovary is primarily composed. I do not consider my own observations on the human embryo sufficiently complete to enable me to offer any criticism on the details given with so much precision as Dr. Pflüger has done, of facts observed by him during a long period of time, but these have been quite sufficient to convince me that the ovaries of the human female do at early periods of embryonic life contain tubular or quasi-tubular structures, which are intimately concerned in the development of the Graafian vesicle. Dr. Billroth[1] has come to similar conclusions, and, from the facts observed by him with regard to similar formations in the thyroid, he came to the brilliant induction (which has only become known to me since I had arrived at the results now communicated with regard to the ovary), that there was every probability of similar growths being found in cystic tumours of this organ. Of this induction I believe that the facts now brought forward furnish an experimental proof; and I think, therefore, that I am warranted in expressing the opinion that these tumours of the ovary should be classed with those which originate in other glandular organs, by an abnormal repetition of the processes of development observed in the fœtal condition, recurring with aberrations in the adult.[2] I use the term aberrations because the normal process of secretion in the ovary is attended with the formation of ova, as that of the testicle consists in the production of spermatic cells. I have carefully sought in the smaller cystic formations for any indications of these structures; but though I have occasionally found cells of dimensions equal to that of the early ova, and often reaching $\frac{1}{1000}$ inch in diameter without appearances of fatty degeneration, while in other cases, where the cells were fattily degenerated and

[1] 'Müller's Archiv,' 1856, p. 159.

[2] *Vide* Billroth's "Researches on the Thyroid," already quoted, 'Müller's Archiv,' 1856; also his paper on "Cystic Diseases of the Testicle," in 'Virchow's Archiv,' vol. viii; also 'Rokitansky,' vol. iii, pp. 104, 108, ed. 1855.

floated free, they were even larger than the above measurement, sometimes attaining a size of $\frac{1}{500}$ inch in diameter, yet, except in point of size, no further resemblance to ova was traceable in them. I was at first strongly inclined to believe that they might represent an immature or imperfect production of ova; but as I have never been able to find in them anything analogous to the distribution of the yolk-granules, or to the Zona Pellucida, nor in those containing large numbers of fat-granules, any trace of the germinal vesicle, I could not exclude the possibility of their being cells derived from the lining membrane, and swollen by imbibition, particularly as in many cases various stages in size could be traced between those lining and still adhering to the cyst-wall and the larger ones which floated free[1] (*vide* fig. 2, *c*, *d*).

The process of the formation of cysts by the constriction of long tubular processes, as I have described it, is an exact counterpart of that described by Pflüger in his account of the origin of the Graafian follicles, while the cystic formations in the glandular growths on the surface have their counterparts in the whole series of follicular glands, of some of which I have given a description in a previous communication to this Society.[2]

The only mode of cyst-formation which I have here described, and which does not come precisely within this category, is that which takes place in connection with the cauliflower excrescences found in the interior of these cysts. It is not, however, so widely different from some of the modes of formation of cysts from glandular structures as to invalidate the general law of these growths. It is, further,

[1] Since this paper was communicated to the Society similar observations have been made by Drs. Woodham Webb and Charles G. Ritchie. These gentlemen appear to consider that the evidence of these large cells, representing "imperfect ova," is more complete than my own observations seemed to warrant me in concluding. *Vide* 'Med. Times and Gazette,' Aug. 6th, 1864.

[2] "Contributions to the Pathology of the Glandular Structures of the Stomach;" 'Med.-Chir. Trans.,' vol. xli, 1858.

somewhat exceptional; and, as I remarked before, the cysts produced by this means do not appear to be very numerous; and some of them, as seen in fig. 42, are apparently subject to multiplication by constriction and division in a manner analogous to the other forms. In one respect they still maintain the glandular type, for they are not formed *within* solid structures, but external to them, and in connection with a glandular secreting surface, as these cauliflower and dentritic growths, equally with the Haversian fringes of synovial membranes,[1] must, I believe, be regarded as secreting surfaces, and equivalent to everted crypts or follicles.

I have not been able, while making these observations, to obtain any instances of multilocular tumours containing dermic structures. As, however, the presence of glands, both of the sebaceous and sudoriparous type, has been demonstrated in them both by Steinlin (l. c.) and by Kohlrausch,[2] I see no difficulty in entertaining the belief that in the cases where they occur, and where they must be regarded as the analogues, by a further aberration in development, of the structures which I have described, they will be found to be the origin of the secondary cysts found in connection with them, more particularly as these dermic glands have been proved by Sir A. Cooper,[3] Wernher,[3] Förster,[4] and Remak,[5] to be not unfrequently the source of cyst-formations of very complex structure. So that in these cases, also, the process will be in its essential characters similar to those which I have described.

With regard to the determining cause of this remarkable condition of growth and development, we must, I believe, as yet confess ourselves almost entirely in ignorance. Führer's[6] theory, founded on some facts which have come

[1] 'Quain and Sharpey's Anatomy,' ed. 1848, p. 301.
[2] 'Müller's Archiv,' 1843.
[3] Quoted in 'Paget's Lectures,' pp. 436, 437.
[4] L. c., vol. i, p. 191.
[5] 'Deutsche Klinik,' 1854, p. 170.
[6] Ibid., 1852, pp. 200-1.

under his observation, that the disease commences with obstructions to the Fallopian tubes, has, as he well points out, certain analogies in its support from similar conditions occasionally observed in the mamma; but the fact that in many cases of ovarian tumours the Fallopian tubes can still be found patent renders it difficult to accept this explanation as universally applicable.

It appears to me that the term "degeneration," which has occasionally been applied to these affections, is one which very inadequately expresses the true nature of the pathological process involved, and that it would be therefore better to restrict ourselves to the nomenclature more ordinarily employed, especially as the divisions of the cystic growths into "simple," "multiple," and "multilocular" or "compound," fully expresses their nature and chief characteristics.

In conclusion, I wish to add a few remarks on the mode of investigation which I have pursued in these researches. I have invariably examined all the preparations which have come under my notice by means of sections made in the fresh condition by a Valentin's knife, carried perpendicularly through the inner wall. It was by this means that I first discovered the true nature of the glandular structures which I have described. They can, however, be examined with much greater accuracy; (and this remark applies especially to the small cysts of secondary formation in the wall and stroma,) after being hardened for a few weeks in a solution containing 2 per cent. of chromic acid. Very fine sections can then be obtained; and, on the addition of Liq. Potassæ and glycerine, the structures, including even the nuclei of the epithelial cells, are seen to be entirely unchanged. The same method is also one that I can strongly recommend for the investigation of the cauliflower growths. I have, in the description of the drawings, in all cases stated the mode of preparation employed.

DESCRIPTION OF PLATES VIII, IX, X.

PLATE VIII.

Fig. 1, A, B. Represents nuclei and fibres of the inner layers of the stroma of a parent cyst.

At B the nuclei are seen with one or more nucleoli. Nuclei measure $\frac{1}{1600} \times \frac{1}{2000}$ inch, nucleoli from $\frac{1}{7000}$ to $\frac{1}{3000}$ inch. Chromic acid prep. × 1200 diam.

C. Nucleated cells from wall of a thin-walled cyst. One is seen with two nuclei; all have a nucleolus. Length of cells from $\frac{1}{250}$ to $\frac{1}{120}$ inch. Nuclei average $\frac{1}{2250} \times \frac{1}{7500}$ inch. Recent prep. × 690 diam.

Fig. 2, A. Stratified layer of cells on interior of walls of "parent" cyst; the superficial layers are columnar; the deeper, polygonal.

At *a* a single cell of the superficial layer is seen, with two nuclei.

B. Vertical section through wall of ovarian cyst, where the epithelium was flattened. The nuclei of the fibres beneath are seen assuming a rounded form.

b, b. Show single cells removed by scraping from the upper layer. Recent prep. × 460 diam.

C. The ordinary character of rounded cells in apposition, or separated by a small amount of intercellular substance, which forms the lining of an ovarian cyst. Cells = $\frac{1}{3500}$, nucleus $\frac{1}{2000}$ inch in diameter. Recent prep. × 460 diam.

D, D. Large round cells from surface of smaller papillary growths. Some with one, some with more nuclei.

At *d* the nuclei are seen free, and in a state of fatty degeneration. Size of cells averages from $\frac{1}{1700}$ to $\frac{1}{1500}$ inch, of nuclei $\frac{1}{2250}$ inch. Recent prep. × 460 diam.

E. Lining of cells in close apposition which could be stripped as a membrane from a thin-walled cyst. Average diameter of cells $\frac{1}{1400}$ inch, of nuclei $\frac{1}{2250}$ inch. Recent prep. × 460 diam.

Fig. 3. Represents the commencing production of secondary cysts from Graafian follicles. In both *a* and *b* a narrow tubular process is seen connecting two follicles. It is a question whether two adjacent follicles have thus opened into one another, or whether one is a dilatation of a process given off from the others. The

PLATE VIII—(*continued*).

former view appears to me the most probable. The ovary was studded with microscopic cysts, showing every gradation in size, from that of a Graafian follicle upwards, varying from $\frac{1}{150}$ inch diameter upwards. With the exception of the figure here drawn, these were all distinct. Chromic acid prep. × 250 diam.

Figs. 4 to 15. Represent the structure of cauliflower growths, and the modes in which cysts are formed in them.

Fig. 4. Represents a series of small papillary growths, covered with epithelium, springing from the wall of a parent cyst.

a and *b*. Represent the earliest stages in which they assume a definite shape. They are seen by their irregular enlargements, and by overlying the wall of the parent cyst, partially to enclose spaces as *c*, *c*, *c*, covered with the same epithelium as the wall of the parent cyst. A loop of vessels was seen to shoot up into each.

Fig. 5. Represents at *g*, *g*, large rounded masses, formed by the enlargement of such growths as *a*, *b*, figs. 4 and 5. From these masses secondary small growths (*d*, *d*) are springing, having the same characters as *a*, *b*. They project into the cavity of the cyst, and give the larger masses a velvety appearance.

Between these masses are seen, at *e*, *e*, *f*, irregular spaces, already partially enclosed by the growth of the excrescences.

Fig. 6. Shows a view in profile of the mode in which the secondary dendritic growths spring from the parent stem. These are seen at *b*, *b*. Spaces bounded by the growths and by the wall of the parent cyst are seen already partially enclosed.

At *d*, *e*, similar spaces are seen to be forming between the secondary growths on the larger mass.

At *e'*, is seen a cyst formed in the wall by such a continuation of this process as is represented at figs. 7 and 8. Chromic acid prep. × 100 diam.

Fig. 7. Represents sections of two growths which have sprung by narrow peduncles (*a*, *a*), from the wall of the parent cyst. These have given off mucous offshoots in very irregular directions. Some of the offshoots have bent down, and again come in contact with the wall of the cyst, thus giving rise to the irregular spaces *c*, *c*, *b*, *b*, which latter are already almost enclosed. Chromic acid prep. × 100 diam.

PLATE VIII—(*continued*).

Fig. 8. Represents a section through four growths (*f, f, f, f*), the adjacent walls of which have met superiorly, and enclosed the spaces *a, a', c.* From *a'* and *c* prolongations of these processes have been formed as described in the last, and continued into the stroma.

At *e* another, which had apparently been previously in connection with *c*, is almost completely separated as a cyst; and the same may be seen with respect to the connection between *d* and *a*. Chromic acid prep. × 250 diam.

Fig. 9. Represents a section through portions of three or four growths, slender and pedunculated, which have united above, and left inferiorly the spaces *d, e, e*, still covered by epithelium. Above, similar spaces are also left at *a, b, c*, in all stages of more or less complete conversion into closed cavities. Chromic acid prep. × 250 diam.

Fig. 10. Represents a section of a portion broken off, and free in the field of the microscope, from the end of one of these cauliflower growths. It is seen to be giving off two offshoots, and in its interior are seen two spaces (*a, a*), both lined by epithelium, and produced in the manner last described. Chromic acid prep. × 250 diam.

Fig. 11. Represents the stroma of one of these masses.

a. Showing at their free extremities a delicate hyaline, finely striated structure, enclosing a few nuclei; deeper in the tissue the stroma is more striated, and lines of nuclei mark the course of the vessels. The more fibrous tissue passes by insensible gradations into the comparatively structureless extremities of the growths.

b. Represents the stroma of a commencing growth. Both *a* and *b* have lost their epithelium. Recent prep., without reagents, × 460 diam.

Fig. 12. The end of one of these growths magnified 250 diam. Recent prep. In some cases, where the growth is very rapid, the epithelium attains a very large size, as seen in fig. 2, *d, d*.

Figs. 13 and 14. Show the commencement of one of these growths from the cells of the connective tissue. It is probable that there is a great increase at a later stage of the intercellular substance, in which the cell-walls become merged, while the nuclei of the cells represent the nuclei seen in the delicate stroma of the growth. At first the cells are closely packed, and lie thick together. Recent prep., without reagents, × 460 diam.

PLATE VIII—(*continued*).

Fig. 15. Represents the trabecular appearance seen on a vertical section with a Valentin's knife through one of the larger masses of cauliflower growth. The irregular growth, enlargement, and coalescence of the papillæ form a series of spaces lined by epithelium, and often communicating with one another. The meshes contain large vessels. These do not appear to have dilated as cysts, nor do they in most cases form completely closed cavities.

PLATE IX.

Figs. 16 to 35. Represent the growths of villi, tubular glands, and cystic formations from the glands.

Fig. 16. Represents three scattered villous growths covered with columnar epithelium. Some are slightly club-shaped, others are pointed. At *a* one is seen denuded of its epithelium; they are then seen to contain a loop of vessels, supported by a very delicate connective tissue, in which are some scattered nuclei. Recent prep. × 250 diam., without reagents.

Fig. 17. Represents the commencement of the formation of glands. Into the stratified mass of cells covering the surface spring up short papillæ, carrying vessels and a small amount of connective tissue; the spaces between these are gradually surrounded by a further growth inwards of the stroma of the walls of the cyst (*vide* text, pp. 254-5). Chromic acid prep. × 250 diam.

Fig. 18. Represents a gland-mass of epithelium stripped from its containing follicle; part of the follicle has probably been torn away with the epithelial mass (*a*), and tends to holds the cells together. The papillæ between which it lies are then seen at *b*, *b*.

At *c* is the fossa left by the epithelium, up the sides of which the connective tissue was extending. Chromic acid prep. × 250 diam.

Fig. 19. Two very long villous growths, which reached almost entirely across a small cyst. They show numerous sinuses or diverticula, formed by inflections of the villi. (I do not think it possible that these could have been artificially made. The cyst was not compressed, and the section was made with a Valentin's knife.) Chromic acid prep. × 250 diam.

PLATE IX—(*continued*).

Fig. 20. Vertical section of a glandular growth imbedded in the stroma. They are not diverticula from a uniform surface, but their ends project as villi. Loops of vessels are seen crossing them at various heights, showing that they are completely imbedded. Chromic acid prep. × 250 diam.

Fig. 21. A glandular growth proceeding with great rapidity in the interior of a secondary cyst. Two glands are seen in process of multiplication by division, owing to the growth upwards of septa from their expanded bases (*a*, *a*).

At *b* the occlusion of the orifice of one of these glands by the pressure from adjacent growths. (Some of these, for distinctness' sake, have not been drawn.) The two sides have grown together, and a further growth is proceeding at the end.

At *c* a cyst, formed probably in a similar way, and the origin of which could be traced in a long fibrous cord proceeding from the side turned to the inner surface of the cyst (not figured), is already separated from the glands by the growth of the stroma and the distension of the cyst-wall. Chromic acid prep. × 150 diam.

Fig. 22. A large tubular process, with thickened walls, probably resulting from a dilated gland, has a septum forming across it, dividing it into two cavities. Chromic acid prep. × 250 diam.

Fig. 23. A secondary cyst, partially filled with a gland-growth, and representing all the processes of gland formation.

At *d* the cyst-wall is seen to be lined with only a single layer of epithelium. At *e* this has become stratified. At *f* papillæ are seen in course of growth; and at *g*, *g*, *g*, the further growth of the glands is seen. At *a*, *a*, are seen the division of the base of the glands by septa projecting from the base; at *b* the commencing closure of the orifice of one of the glands by the pressure of those adjoining; at *b'* a gland, multiple at its base, has been converted into a cyst in the same manner; *h*, *h*, are cysts which have had a similar origin. The ends of the tubes from which they were formed have continued to grow and branch as septa (*c*, *c*, *c*) within the cyst. Chromic acid prep. × 150, somewhat reduced.

Figs. 24 and 25. "Cysto-sarcoma," or "alveolar degeneration" of the ovary, produced by the gradual thickening of the walls of such a series of glands as partially fill the cysts figured in the last. The figure represents a section across a series of dilated tubular

PLATE IX—(*continued*).

follicles, many of which are divided by imperfect septa, and also lined with epithelium. The stroma and septa were highly injected. The spaces measured from $\frac{1}{40}$ to $\frac{1}{15}$ inch in diameter. Fig. 24 × 100 diam. Fig. 25 is a portion of fig. 24 × 250 diam. Recent prep.

Fig. 26. Represents the first stage in which the glands formed on the interior of the parent cyst become encapsuled within its wall. The stroma has already partially grown over their orifices, and a fresh growth of epithelium and papillæ is appearing on the surface.

At *a*, *a*, are seen two diverticula from one of the glands.

At *b* is seeh a gland almost closed superiorly by the pressure of adjacent growths; it is partially divided at its base, where a cavity is already in course of formation. Chromic acid prep. × 150 diam.

Fig. 27. Represents a process by which successive strata of glands may be encapsuled in the wall. The growth of the stroma is in such cases irregular, shutting off masses at different times. The septa between the glands widen, so that masses are shut off, separated by wider interspaces of stroma than intervened between them in the first instance. Sometimes a large compound growth of glands and stroma will overlap and enclose one which has proceeded less rapidly, and these again become, in their turn, overgrown by the stroma. Many of the glands in this preparation are seen to be divided at their base. Chromic acid prep. × 150 diam.

Figs. 28, 29 (Plate IX), 30, 31 (Plate X), are representations of varieties in the result of this process. In fig. 28 composite masses of glands are seen imbedded below the surface, and are slightly elevated above it as small, hard nodules, of the size of millet or poppy seeds.

Such masses as *a*, *b*, *c*, when expanded into cysts, give rise to compound alveolar forms, wholly or partially separated by septa.

Others, again, as *e* (which fig. 29 represents more highly magnified, to show a repetition of the same process commencing in a secondary cyst), are portions of simple glands, shut off, and which expand side by side with the others, as we find the secondary cysts, when growing, are sometimes unilocular, sometimes multilocular, and that these varieties occur mingled together at fig. 28. One of these has already begun to form a cystic cavity. Chromic acid prep. × 150 diam.

Plate X.

Fig. 30. Is drawn to represent a cyst formed in the manner last described, which still gave evidence of its origin by a long tubular process proceeding from its upper side, and losing itself among the new growths of glands and stroma which had covered over the original growth.

a. The cyst, with a secondary formation of stratified epithelium commencing in its interior.

b. The tubular process leading from it. Chromic acid prep. × 150 diam.

Fig. 31. Gives an illustration of the same mode of proof of the origin of these cysts, except that it is shown in a less distinct manner at *b*, where a fibrous cord, extending from the upper part of the single cyst to the superficial layers of the stroma, marks its mode of origin.

At *a* the tubular origin is more distinctly marked. The upper surface, in both this preparation and the last, is covered with a new growth of epithelium and villi. Chromic acid prep. × 150 diam.

Fig. 32. Represents a condition in which these glands, deeply imbedded in the wall, are often found (in a quiescent state?—*vide* text, p. 260). They show no disposition to dilate into cavities, but appear to possess at any time the power of doing so. (Qy. Is this the condition described by Professor Förster?)

Fig. 33. Represents a condition which I believe to result from the fatty degeneration of the cells of the glands, as represented in fig. 32. They correspond precisely to these masses in form and arrangement, are found in the same parts, and often in juxtaposition in the same tumour. They appear to give rise to the next two conditions depicted.

Figs. 34 and 35. Are, I believe, conditions which are the result of the fatty degeneration of these glands last described. The oil-globules are absorbed, or the stroma undergoes the same change, and breaks down in part, and a series of irregular areolar spaces are left in the stroma, some still persisting as closed cavities, others communicating with one another and with the surfaces, but all destitute of an epithelial lining. This may take place to a much more marked extent than has been figured here. I believe that such structures may have given rise in part to the theory of the alveolar origin of these cystic formations.

PLATE X—(*continued*).

Fig. 36. Diverticula from wall of a thin-walled cyst, opening into its cavity by narrow orifices, and expanding on the outer side into large, hollow, flask-shaped processes, lined by a spheroidal epithelium. Their lining membrane was partially thrown into folds from the pressure of the covering-glass. Natural prep. with glycerine, × 100 diam.

Figs. 37 and 38. Instances of small cysts giving off diverticula, which only become hollow after they have gained considerably in length. They have no membrana limitans. In fig. 38 is seen a process of secondary cyst-formation from one of these diverticula by a process of constriction.

At *a* a cyst is already formed in this manner, and the process is seen commencing at *b*. A similar process appears to be commencing at fig. 37, *a*. Both are chromic acid preps. Fig. 38 × 150 diam., fig. 39 × 250 diam.

Fig. 39. Represents a long tubular process passing from a small dilated cyst (A). The narrower part (*a*) has complete constrictions at *b*, *b'*; *b*, *c'*; *c*, *c'*; *c'*, *d*; at *d* it passes into a long process, in which at first two and later only a single row of columnar cells are visible. There was seen, on altering the focus, another constriction between A and *b*, but this could not be drawn.

At *b*, on altering the focus, a partial continuity of the epithelium through the constriction could also be seen. There is another partial constriction between *b* and *c*. Chromic acid prep. × 230 diam., reduced.

Fig. 40. The end of a crypt similar to those last described, seen with a superficial focus. There is observed a crowd of nuclei where the epithelium ends, and where the stroma does not surround the end of the crypt so closely as in other parts. Further removed from the end of the tube the nuclei are seen more elongated. Chromic acid prep. × 460 diam.

Fig. 41. Portion of an alveolar arrangement of the cysts, forming a less-marked condition of fig. 24 (Plate IX), where the septa of the cysts were in a condition of fatty degeneration. The epithelium still preserved; but at *a* are seen ragged remains of cyst-walls which have ruptured. Part of the wall of the larger cavity is still smooth, but at other parts the growth of septa beginning as papillary processes may be seen extending into it, and these are seen repeated in some of the smaller cavities. Many of these were seen, under higher powers, to contain glandular

PLATE X—(*continued*).

structures, similar to those in figs. 20, 23 (Plate IX). Others contained large villous growths, as in fig. 19. This figure was taken from another portion of the same tumour as that from which fig. 24 was drawn. Chromic acid prep. × 74 diam.

Fig. 42. Gives a further illustration of the multiplication of cysts by division.

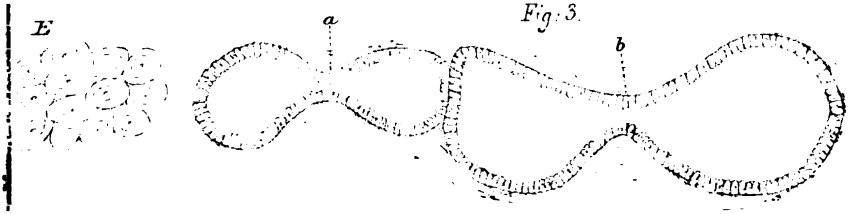
c. Was a small cyst of the size of a pin's point, to the naked eye, seen through the peritoneal covering of one of the cysts containing papillary growths before described. On making a vertical section through it and the wall below with a Valentin's knife, I found the structures as here figured.

a. Represents the inner wall of the parent cavity, covered with small papillary and commencing cauliflower excrescences.

b. Represents the peritoneal surface, which appeared almost to form the outer wall of the cyst *c*.

Between *c* and the inner wall lay a long, double, club-shaped cyst (*e*), which was separated from *c* by the blood-vessel *d*. It presented a constriction at its centre (*h*); and across this, and partly enclosing it, was a band of thick fibrous tissue (*f*) which was spread into and lost in the rest of the stroma. My impression is that the cyst *c* had been formed by the earlier constriction of a portion from *e*, and that *e* had been formed from one of the papillary growths in the manner before described. They were both lined with polygonal cells, precisely similar in both cases, of which *g* is a specimen, as seen in *c*, magnified 460 diam. Recent prep. × 100 diam.

Fig. 3.



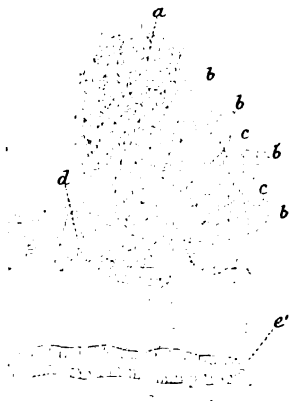


Fig. 7.

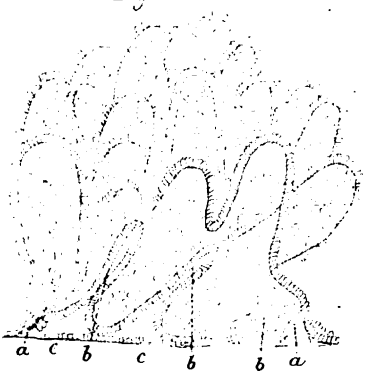


Fig. 11.

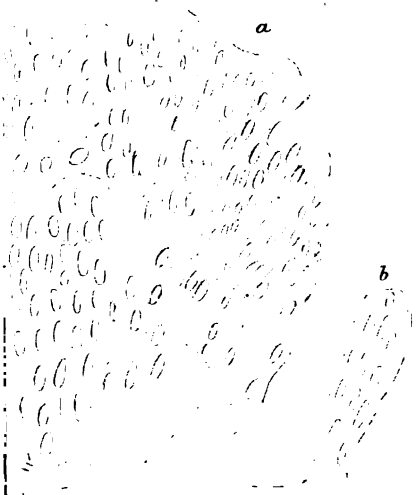


Fig. 13.

Fig. 14.

Fig. 15.

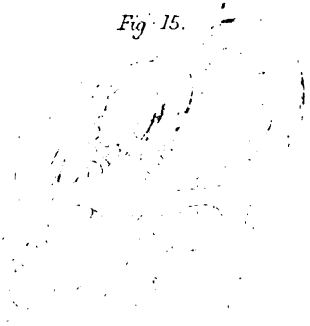


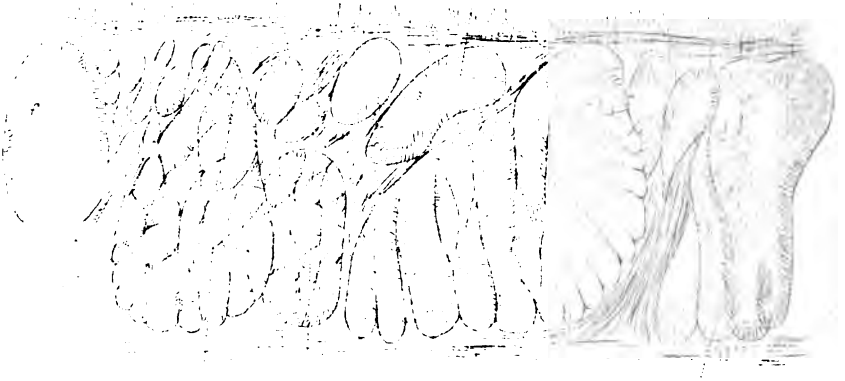
Fig. 19.

Fig. 20.

Fig. 22.

Fig. 29.

Fig. 28.



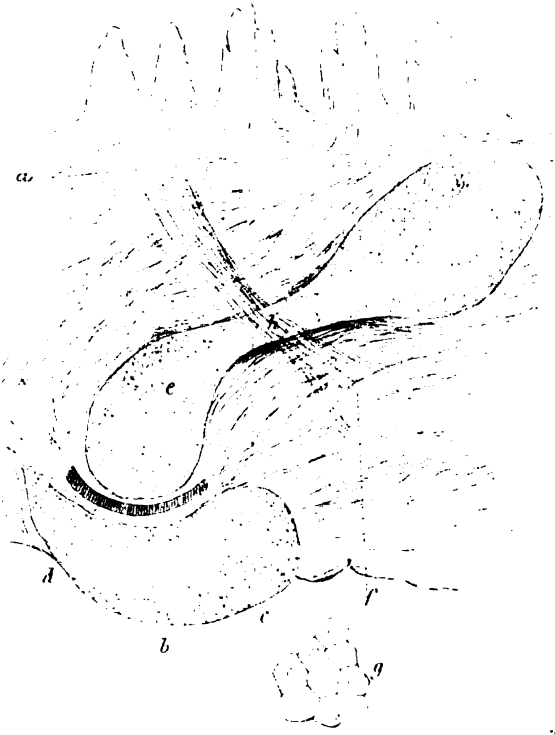
b c a

W. West. imp

Fig. 41.

A

Fig. 42.



A SUCCESSFUL CASE

OF

PARACENTESIS CAPITIS.

BY

THOMAS YOUNG THOMPSON, M.R.C.S., L.S.A.,

NEWCASTLE-ON-TYNE.

COMMUNICATED BY

DR. FULLER, HON. SEC.

Received June 18th.—Read June 28th, 1864.

I BRING the following case before you as an example of the value of an operation by some considered inadvisable, and of which the successful cases recorded are few.

On June 8th, 1861, I attended Mrs. M— of a fine healthy boy. When fourteen days old the child sustained a fall, apparently without any ill effects; but at the end of three weeks a protuberance appeared on the crown of the head, at first circumscribed, but gradually becoming diffused.

The head then began slowly to enlarge, and on the 3rd August, 1861, my opinion was asked. The child was two months old, well nourished, noticed surrounding objects, and slept well. His features were not expressive of pain,

there was no strabismus, no twitching of the limbs, and no convulsions, or other indication of irritation of the nervous centres. The head was very large in comparison with the body, which was well developed and of ordinary size. The enlargement was general, and uniform; fontanelles not protruding; sutures all widely separated, especially the coronal and sagittal. The eyeballs more prominent than natural, the upper part of the globe only visible, the lower lid covering the two lower thirds, even beyond the pupil. When the child's attention was attracted it threw back its head, and by this means, no doubt, was enabled to see, the pupil becoming then uncovered. When the head was placed between the observer and a candle it was perfectly transparent. This was most perfect when viewed from side to side, and even anteriorly through the orbits and occiput.

At this time the tincture of iodine was applied each day to the scalp, and a grain of calomel was given twice a day. This treatment was continued until the 15th August, without any improvement, when a grain of iodide of potassium was substituted for the calomel.

A gradual increase of the head occurred, and on September 25th, 1861, it measured, circularly, the tape being placed over the parietal eminences, twenty inches, and laterally, encircling the chin, twenty-one inches.

At this time cod-liver oil and the phosphates of iron were commenced, with occasionally the application of the croton-oil liniment to the scalp.

April 28th, 1862.—An improvement is perceptible in the condition of the child's health, but the head continues to increase, and now measures, circularly, twenty-four and a half inches, and laterally twenty-four and a quarter inches, the head now increasing at the rate of an inch in two weeks. It falls to the side upon either shoulder.

The frontal bone projects greatly, and the parietal bones are widely separated. The surface is very tense, and fluctuates over a space fully four inches across, and the whole length of the sagittal suture. The face appears very small

in comparison with the head. Its triangular contour is very peculiar.

The intellectual faculties appear to be developed to the same degree as is usual for the age; hearing appears perfect; the limbs are moved well, and the alvine secretions are natural.

At this time I considered that, as the treatment with iodide of potassium, &c., had been continued for ten months without benefit, but, on the contrary, with an increase in the collection of the fluid; as the emaciation was great; as the child was born of healthy parents, without any tubercular history; and as the primary cause of the disease was an accident, the case was one peculiarly suitable for the treatment by paracentesis.

This opinion was fully coincided in by my friends Dr. Heath and Dr. Philipson, who saw the case at this time. The parents of the child also, having been fully warned of the uncertainties that might be expected, willingly sanctioned the proceedings.

May 3rd, 1862.—In the presence and with the kindly assistance of Drs. Heath and Philipson, the child being supported, I carefully selected a spot quite free from superficial veins, on the left side, about one inch and a half from the anterior fontanelle, and introduced a trocar and canula, about the size of an ordinary crow-quill, through the coronal suture. Directly upon the withdrawal of the trocar, a clear, colourless liquid began to flow, of the same consistence as water, and closely resembling it. After about ten ounces had been allowed to flow the canula was withdrawn, and a piece of lint applied. Not a single drop of blood was lost. The head was then firmly encircled in broad strips of adhesive plaster, laterally and circularly. A little weak wine and water was administered, and the child placed in bed, as slight symptoms of faintness were noticed. Upon the withdrawal of the fluid the scalp began gradually to be less tense, more and more concave, and finally exhibited a most singular appearance, the edges of the separated bones being boldly visible, enclosing a hollow space.

The fluid was of sp. gr. 1010, and contained not a trace of albumen. The fluid continued to flow during the whole of the next twenty-four hours, and a few ounces must thus have escaped.

The day after the operation slight febrile symptoms occurred, but upon the administration of a diaphoretic they gradually subsided.

On the seventh day fresh strips of plaster were applied.

On the tenth day, about 2 a.m., a strong convulsion occurred, which was said to have affected the whole body, but when I saw the child, shortly afterwards, the movements were confined to the right leg and arm, contractions alternating with relaxations, the child remaining quite sensible; the gums were very tense, two teeth appearing ready to protrude. I accordingly scarified the gums, and administered an aperient—calomel and castor oil. The movements continued for two hours, then gradually subsided, and finally passed off. They did not again recur.

The scalp gradually became more full, and on the 20th, seventeen days after the operation, the iodide of potassium was again given.

June 9th, 1862.—The head appears nearly as large as before the operation; the child in much the same bodily condition. The operation was performed the second time, and about four ounces of liquid withdrawn. This time it was milky, and of the consistency of weak gum water; no untoward symptoms occurred. The head remained much in the same condition, and on July 25th measured, circularly, twenty-two inches, and laterally twenty-three inches. A change of air to the sea-side was now tried, and, whilst sea-bathing was being employed, cod-liver oil and preparations of iron were again given. The head did not enlarge, but, on the contrary, remained much in the same condition, and at the end of August the soft parts were still depressed; the child's general health was improving. During 1863 this continued, and it was noticed, further, that the scalp was becoming more and more firm.

June 8th, 1864.—The child is now three years old, strong,

and well nourished; the head, in comparison with the body, does not appear so large; it measures, circularly, twenty-three and a half inches, and laterally twenty-three inches. On the vault no soft parts can be felt, the whole surface being firm. There is no indication of any mental impairment; on the contrary, the child is very intelligent; the teeth are all present.

This case is interesting, as it has remained under observation during the *whole period of the disease and the cure.* It shows most forcibly that sometimes the most unpromising cases finally do well, and that this operation may be followed by a successful result.

It is difficult to say where the fluid was situated, or to determine its relation to the brain-tissue. We supposed it was situated in the sac of the arachnoid. This supposition is strengthened by the history of the case, the swelling first appearing circumscribed, and then becoming diffused, and finally being followed by enlargement of the whole head.

After the first operation, in all probability, inflammation occurred, evidenced by the sudden accession of symptoms of irritation and by the appearance of the fluid obtained in the second tapping. This morbid process probably prevented more fluid being exuded or secreted, or, by being of greater consistency, mechanically, by its pressure, caused that which was present to be absorbed.

ON THE

CAUSES OF HERNIA.

BY

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THE apology for reviving so well-worn a subject is the belief that hernial pathology has not yet found its stable equilibrium. The opinions which obtained up to the end of the last century have given place to a more purely mechanical interpretation; and as this interpretation is being made the basis of treatment, it seems important to test its validity. It is with this view that the writer ventures to submit to the Society the following considerations.

The prevailing view of the causes and nature of hernia—that of Scarpa and his followers—may, for convenience, be called the "mechanical;" the other, of which Richter was the most famed exponent, may be called the "pathological." The supporters of the "mechanical theory" assert that the abdominal viscera are maintained in their several positions by the pressure of the muscular parietes, and that the mesenteries, ligaments, and peritoneal folds, are nutritive attachments rather than mechanical supports; that the intestines are normally prone to displacement, and that hernia depends upon a loss of balance between the muscular power of the parietes and the resisting power of the fibrous fabric of the inguino-crural aponeuroses.

The advocates of the "pathological" theory, on the other hand, maintain that the intestines (with certain exceptions, that need not now encumber the argument) are not prone to displacement in the normal and healthy condition of the mesenteries, ligaments, and peritoneal folds. They agree that the protrusion is the result of the mechanical action of the muscular parietes, but hold that the intestines do not come within the sphere of that expulsive action until they have descended somewhat in the cavity of the abdomen, either by relaxation, hypertrophy, or other lengthening of their attaching membranes.

Richter[1] argues that such elongation is a necessary precursor of protrusion. Scarpa,[2] on the contrary, asserts it to be merely a consequence, or, at the least, a concomitant, of protrusion, due to the dragging of the protruded viscus. Malgaigne[3] affirms that inguinal hernia can take place without any lengthening whatever of the mesentery, either previous or concomitant; and there are many recent writers of the same opinion. But there is no need to regard them now.

Scarpa denies the necessity of relaxation simply on the strength of an assertion which he fails to prove. He says that the ligaments of the viscera are not strong enough to support them *in situ*. The fact that the liver, the weightiest of all the viscera, is sometimes lacerated by the resistance of its own ligament under the shock of a suddenly arrested fall, countervails his assertion.

Malgaigne,[4] too, deals very lightly with the same subject. The veriest tyro, he says, knows that the mesentery will allow the intestines to pass beyond the walls of the abdomen; but he forgets to state what part of the walls, or how much of the intestine.

Cloquet[5] rested his elaborate argument on the fact that

[1] French translation, by Rougemont, p. 10.
[2] Translation by Wishart, p. 44.
[3] 'Leçons Cliniques sur les Hernies,' pp. 29, 30.
[4] Op. cit., p. 29.
[5] 'Recherches sur les Causes des Hernies Abdom.,' 1819.

hernia is more frequent on the right side of the body than on the left. Malgaigne[1] showed that hernia on the right side was very frequent in left-handed persons, and *vice versâ*, and so upset his conclusions. Out of 374 male cases of hernia (inguinal), interrogated consecutively as they came under notice at the City of London Truss Society, in the year 1860, 350 were right-handed persons and 24 left-handed. Of the 350 right-handed patients, 201 were ruptured on the right side and 132 on the left, while in 17 the hernia appeared on both sides simultaneously. Of the 24 left-handed, 13 were ruptured on the right side and 11 on the left. This confirms Malgaigne's objection; but the tables[2] of the Truss Society, just mentioned, show more than this. It is true that, in the aggregate of all classes and every age, the right side is more often the seat of hernia than the left (nearly one third); but it is interesting to observe that the age to which Cloquet's arguments are intended to apply, namely, the period of full manhood, happens to be that during which the two sides are more equally affected. During the first few months of life the lesion occurs on the right side three times oftener than on the left. This disparity lessens as puberty and manhood approach. As the bodily powers decline, right ruptures become more frequent. Nor is this all. Hernia occurs on the right side eight times oftener during the first year of life than during any subsequent year; and when the fullest allowance is made for the excess of infant population over the adult,[3] still the first year of life will be found to afford more than four times the number of patients with right ruptures than any subsequent year. It is clear, then, that there must be some other cause than muscular exertion to

[1] 'Leçons Cliniques,' &c., 1841, p. 32.

[2] See Table appended.

[3] The following is obtained from the Census of 1851:

Males under one year . . .	289·371
„ between one and five . .	887·382
„ between twenty-five and thirty .	617·889

Compare this with the Table appended to this paper.

bring about this predominance of right over left hernia. Wrisberg and Camper found an explanation of it in the more tardy closure of the right tunica vaginalis testis. This interpretation is accepted by Malgaigne.[1] It fits in with the tables of the Truss Society, and is generally admitted.

Labour has been thought to favour the "mechanical" theory. The statistics of the Truss Society do not confirm this. During three successive years the occupations of the male patients who came for relief to the City of London Truss Society were carefully noted down, and the list, showing the number of patients of each occupation, has been compared with a list taken from the General Census of 1851.[2] The result is that (with certain exceptions, which enhance

[1] Op. cit., p. 48.

[2] The following is the list alluded to:

TRUSS SOCIETY.

(The society is situated in the heart of the Metropolis.)

ORDER IN CENSUS 1851.	1859.	1860.	1861.
Farm Labourer	—	171	173
General Labourer	*776	†553	†734
Boot and Shoe Maker	38	53	72
Carpenter and Joiner	‡173	‡178	99
Tailor	20	33	28
Domestic Servant (male)	101	176	131
Silk Weaver	63	71	58
Blacksmith	48	51	63
Mason, Pavior	—	18	—
Messenger, Porter, &c.	478	410	351
Gardener	65	119	114
Bricklayer	—	—	49
Butcher	53	52	52
Painter, Plumber, &c.	33	45	50
Baker	35	69	52
Carman	73	87	83
Clerk (Commercial)	29	30	65
Cabinet Maker	—	—	41
Bargeman	—	44	35
Sawyer	35	34	29
Rail Labourer	—	—	—
Hawker	33	57	37
Wheelwright	10	—	18
Printer	36	51	42
Cooper	20	32	23

* Including farm labourers, bricklayers, and rail labourers.
† Including masons, paviors, and rail labourers.
‡ Including cabinet makers.

rather than disparage the conclusion) hernia occurs oftenest in the most numerous classes, and not in the most laborious, except in so far as they are also numerous. The exceptions are shoemakers and tailors. These, being essentially sedentary callings, might be accepted, as favouring the mechanical theory, were it not for the following circumstances:—1st. The fact of the work not being such as to elicit an early manifestation of the malady, even though a predisposition be latent. 2ndly. When produced, the hernia does not so imperatively demand mechanical support; patients of these trades being apt to neglect this protection altogether, or to postpone its adoption. 3rdly. When once a truss has been adjusted it will last three or four times longer than one worn by a labourer who sweats, and therefore so frequent an application to the Society is not needed.

A comparison of the sexes of hernia-patients is also thought to favour the mechanical theory, because males apply for relief so much more frequently than females. But here, again, the arguments are not at all conclusive. In the first place, the disparity is not so great as at first appears. Women make their trusses last more than twice as long as men. They dislike the ordeal of examination and adjustment, and put off their applications for relief till pain or apprehension compel it. Malgaigne,[1] whose statistics are very reliable, calculated the proportion to be four males to one female. The tables of the City of London Truss Society indicate a proportion of five to one. But it is likely that Cloquet's[2] estimate of two to one is the most near the truth, since he deduced it from dissection. The two former were drawn from the patients of charitable institutions; and although in each calculation care was taken that none should be counted a second time, yet, as

[1] 'Leçons Cliniques,' &c., p. 12:

"En 1836, sur 2767 hernies, 2203 hom., 564 fem. = 4 à 1.
„ 1837 „ 2373 „ 1884 „ 489 „ = 3·89 à 1.
" De ces trois colonnes de chiffres il faut conclure le rapport de 4 à 1."

[2] 'Recherches sur les causes, &c., p. 3, note: "Des 457 cas de hernies que j'ai disséqués, 307 appartenaient à des hommes, 150 seulement à des femmes."

they are based upon annual reports, it is needful to make further allowances: for example, if men wear a truss one year, women will wear one nearly three years. It is clear, then, that in an annual report the number of males (even though never counted a second time, of which there must always be some chance) will be unduly swelled in proportion to the other sex. The number of males should be at least halved to express the proportion with any fairness. This would establish Cloquet's estimate. If vaginal enterocele, prolapsus of the pelvic viscera, and umbilical hernia be taken into the account, the disproportion between the two sexes will be still further lessened.

Popular belief, however (which generally has truth for its foundation), is decidedly in favour of the mechanical theory. There can be no question that the prevailing notion is that a "rupture" is the consequence of some violent effort. Under this preconception, patients are apt to attribute the cause to any memorable shock, or violence, next preceding the discovery of the swelling, whether the interval be measured by minutes, days, or months. But the connection is seldom clear. It is not common to obtain clear testimony of violent exertion during the day being followed by discovery of the swelling at night, and still more rare is it to get satisfactory evidence of hernia ensuing as a direct and immediate consequence (without any previous sign) of such violence, and yet this is sometimes, though very rarely, afforded. For instance, when the tunica vaginalis testis[1] remains patulent, hernia has been said to occur suddenly and fill the scrotum without the patient having had any previous sign or warning of it. Some cases of direct hernia have been known to descend rapidly into the scrotum, but not with the suddenness of the so-called "congenital hernia." Cases of this kind are so rare as to be quite exceptional; and, even if they were not so, they could not be quoted against the pathological theory, but rather in its favour, as will be seen hereafter.

[1] *i. e.* The vaginal process of the peritoneum.

The great majority of patients never pretend to give any definite account of the mode in which their hernia was produced, although they often volunteer a theory. Out of 700 adult males, interrogated consecutively as they presented themselves at the City of London Truss Society, only 48 attempted to assign a special cause. Cough is the cause of its *discovery* in most cases. Many learn their condition for the first time from medical examination. Women not rarely come with irreducible femoral epiplocele, the existence of which has just been made known to them by their "doctor;" sometimes they will confess that they know nothing about its formation, and sometimes they insist that it was produced by some quite recent exertion, accompanied with pain at the spot. Indeed, the patients' accounts of the causes are generally so vague and so various that it has been found impossible to tabulate them. Effect is sometimes given as a cause—the retching and vomiting of strangulation, for instance. "Sneezing" has been accused of producing it, and even "stooping to wash a child's head." Rising from bed, after a protracted illness, has several times been given as a cause. In fact, so far as the investigations upon which this paper is based are concerned, there is no evidence whatever to show that muscular exertion in health, and by itself, is ever a cause of hernia.

When the essential conditions exist, muscular effort unquestionably effects protrusion. Of this there can be no doubt. All that is claimed in this paper is, that the part protruded must first come within grasp of the protruding power; and that in the normal and healthy condition of the peritoneum in its entirety the viscera do not come within such grasp.

If it were otherwise, if the intestines were always in a condition ready for protrusion as soon as a fault in the parietes allowed it, it must of necessity follow that whenever the tunica vaginalis remains sufficiently patulent hernia must invariably occur. But it is well known that this is not invariably the case.[1] In the autumn of 1859 Mr.

[1] A gamekeeper, æt. 24, applied to the Truss Society with hydrocele

Stanley removed a cancerous testicle from a patient in St. Bartholomew's Hospital. During the operation it was found that the tunica vaginalis and the peritoneum formed together one continuous cavity. The communication was free enough to admit the finger easily within the serous cavity of the abdomen. Yet there had never been any hernia before the operation; nor, when the man left the hospital with a sound cicatrix, was there any unusual impulse to be felt at the ring. Such cases, though rare, are not unknown to surgeons.

Among females, too, the diverticula of Nuck are acknowledged, even by Malgaigne,[1] to be as often open as the tunica vaginalis, yet hernia is less frequent. Mr. Lawrence[2] says of them, "it has not been ascertained that these diverticula become closed, . . . nor have we reason to believe that their presence favours the occurrence of hernia."

And then, again, if the mechanical theory were the true and sufficient explanation, we should naturally expect to find that a hernia, once formed, would go on increasing in magnitude indefinitely. But this is not so. Hernia will sometimes remain for years stationary, just beyond the outer ring, without any mechanical support, and only begin to inconvenience the patient when some debilitating illness supervenes. Then at once it will begin to descend into the scrotum, and go on increasing to an indefinite size, and then for the first time awaken the patient to a sense of its importance. It is clear, then, that parietal integrity is not the only hindrance to the descent of the viscera.

The opinion of surgical writers (almost without exception),

and ascites. The hydrocele had been mistaken for hernia, because it disappeared when the patient was recumbent. There could be no doubt that the fluid in the two cavities was the same, because fluctuation was distinctly communicated. The two swellings (of abdomen and scrotum) appeared together a few months before his application. There was no previous history of hernia. The tunica vaginalis, in this case, no doubt formed one continuous cavity with the peritoneum.

[1] 'Leçons Cliniques,' p. 14.

[2] 'Lawrence on Ruptures,' 5th edit., p. 578.

up to the time of Scarpa, was that the mesenteries and suspensory ligaments of the viscera constituted that hindrance, and there is much to favour this view. The altered contour of the belly in ruptured persons favours it—the unnatural fulness of the iliac fossæ, with sinking of the epigastrium and prominence of the hypogastric region. This deviation from the normal figure of the abdomen is the rule in patients of adult life. It seems to be less pronounced in cases of (so-called) congenital hernia, *i. e.* hernia into the tunica-vaginalis.

Rokitansky[1] distinctly attributes internal hernia to a "long, flabby mesentery." He purposely avoids treating of external hernia as belonging exclusively to surgical writers; but he evidently associates the two. And it is worthy of remark, that internal strangulation seems to be associated with a family tendency to hernia, a coincidence suggestive of more than accidental affinity between the two forms.

When the cæcum constitutes the hernia it is manifest that its attaching membrane must have been excessive prior to its protrusion, as it is not possible for any force of the muscular parietes to dislocate it from its place in the normal disposition of its peritoneal covering.

The sigmoid flexure of the colon is less regular in its attachment; it is sometimes of considerable length, and has a mesentery of its own; but this condition is so far from being common that, when it forms the contents of a hernia, its protrusion may fairly be said to be due to an unusual length of its attaching membrane.

But the small intestines, it is argued, are subject to no such restrictions. Their mesentery allows them so free range in the abdominal cavity as to permit their ready escape without stretching. This is said to be demonstrable by experiment. It is partly true. The lower coils of the small intestines do unquestionably occupy the pelvic basin, in the empty state of the bladder, and lie against the abdominal parietes in the inguino-crural regions. But it is only a limited portion of

[1] Sydenham Society's transl., vol. ii, p. 52.

the small intestines that has this reach. The major part hangs higher in the abdominal cavity, above the lower edge of the associated muscles. Nor does it appear that there is any force available to alter their position.

Nestled in amidst the large intestines and the stomach, they hang from the lumbar prominence of the spine (the stable centre of every movement of the body), almost uninfluenced by attitude in their relation to the inguinal and crural openings.

Their position determines the curve of the abdominal muscles, and so directs the resultant of their forces. So long as that is parallel with the radius of the mesentery, the intestines are secure from displacement from that source; and whatever downward pressure can reach them must first pass through the gas contained in the ample chambers of the larger gut.

If, however, some such hypothesis as Richter's be accepted —which presupposes laxity or elongation of the mesentery, together with general laxity of the peritoneum—all the difficulties will disappear. The subject will then come within the scope of Scarpa's argument. By descending in the abdominal cavity the small intestines will alter the contour of the parietal muscles, and change the focus of their forces. The long fibres of the transversalis will ply above them, and drag on the mesentery. The weight of the intestines, apart from the force of parietal compression, will fall on the inguino-crural regions, the most dependent part of the abdomen. Every jolt and motion of the body will add to the pressure. Then will occur that loss of equilibrium so much emphasised by Scarpa and Cloquet, and it will become almost a matter of necessity that, sooner or later, the weakest part must yield. Under these circumstances any unusual exertions may act as "the last feather," and either, in reality, bring about the protrusion, or, at least, so far increase it as to be credited with the entire blame.

But it is not only in its mesenteric duplicatures that a laxity of the peritoneum leads to hernia. Its parietal layer

determines the species by directing the channel of escape. It used to be said that the chief "predisposing cause"[1] of hernia lay in the naturally greater size of the openings at which they protrude, "since in males, where the abdominal ring is naturally capacious, inguinal herniæ occur in great numbers, while the femoral species is rare; females, on the contrary, having the capacity of these apertures reversed, are seldom affected with inguinal hernia." But there is fallacy in this.

In the first place, it is clear that the size of the aperture (*i. e.*, the fibrous aperture, for the argument supposes there is no real opening before protrusion) cannot cause the lesion, since it has been shown that an opening may remain permanently gaping to receive one, and no protrusion pass. The condition of the aperture may determine the species, but it cannot produce the complaint.

In the second place, it is not a fact that "the inguinal variety is rare in females." The two varieties (namely, inguinal and femoral) are nearly equal. Malgaigne thinks the inguinal form the more frequent.[2] The tables of the City of London Truss Society make femoral hernia rather exceed the other.[3] But Malgaigne's estimate is probably

[1] 'Lawrence on Rup.,' 1st ed., 1807, p. 7; and 5th ed., p. 44; 'Teale on Abd. Hern.,' 1846, p. 40 and 41; Cloquet, and others.

[2] "J'etais, et je suis encore d'avis que les hernies inguinales sont plus communes chez les femmes que les crurales." 'Leçons Cliniques,' p. 174.

[3] The following condensed analysis of the female patients personally examined at the Truss Society during the year 1863 fairly represents the average of four years' tables:

	INGUINAL.					FEMORAL.				
	Right	Left.	Double.	Total.	With Family History.	Right	Left.	Double.	Total.	With Family History.
Under age of 15...	59	44	13	116	44	7	1	—	8	2
Single women.....	38	23	7	68	28	34	30	6	70	20
Married women never pregnant	8	5	2	15	6	10	5	2	17	4
After miscarriage .	—	—	—	—	—	2	—	1	3	1
Mothers	71	98	29	198	65	182	93	38	314	91
	176	170	51	397	143	235	129	47	412	118

correct. This, however, is a matter of very small importance, so long as it be conceded that females are equally liable to either kind. So that, if the reasoning be sound, the argument defeats itself through the inaccuracy of its data. It proves that the size of the fibrous apertures, not only does not predispose to hernia, but does not even determine the variety. It proves, by inference, that there must be some other regulating cause. This will be found in the parietal layer of the peritoneum. When the protrusion takes place through an unclosed diverticulum of Nuck, the species of hernia will be due to an abnormal arrangement of the parietal peritoneum. The same will be conceded for all cases of true congenital hernia, and whenever the tunica vaginalis testis forms the sac. But there is further evidence than this to show that the lining membrane has more to do, than have the fibrous apertures, in fixing the site of a hernial issue. Among females the kind of hernia depends partly upon age and partly upon sexual condition. The tables of the Truss Society show that, although the two kinds are equal in females, femoral hernia is so rare as to be almost unknown before menstruation. Only four cases have, as yet, come under the notice of the writer.[1] Between the ages of fifteen and twenty-five inguinal hernia still continues to be the more frequent form. After this period femoral hernia becomes the rule. Again, femoral hernia is much more common than inguinal among women who have borne children. Among single women the two forms are more nearly equal. But here, also, the age at which the hernia first appears seems to have more influence in determining the variety, than any other condition. It is found[2] that femoral hernia occurs oftener at an early age in boys than in girls; and yet the femoral opening is not larger in boys than in girls, nor is there any reason to believe that the relative size of the apertures above and below the crural arch becomes altered after puberty. There is, however,

[1] During six years at the City of London Truss Society.

[2] Taking an average of four years.

fair reason to conjecture that the pelvic peritoneum becomes altered in its nature and condition after this period, and partakes of the changes incident to the tissues and organs involved in the function of procreation. It awaits distension as its natural destiny. A certain measure of relaxation or relaxability may therefore be assumed which would favour protrusion in that region, should other conditions exist. And this is probably the interpretation of the fact that femoral hernia is so much more common (proportionally to inguinal) in females than in males.

A young woman, æt. 23, one of the patients relieved at the City of London Truss Society in the year 1860, presented herself with femoral hernia on the right side. Her statement was that she had been ruptured since the age of four years. She was not conscious that any change had taken place in the position of the hernia since its first appearance. The case was therefore looked upon as an unusual example of infantile femoral hernia in a girl. But the books of the society showed that the hernia was not originally femoral. The patient's name was found entered as far back as the year 1844. This and every subsequent entry was in the handwriting of Mr. J. C. Taunton, the then surgeon to the society. Her age then was seven. The nature of the hernia, inguinal on the right side. The next entry was in 1847, age ten, hernia inguinal on the right side. Again 1852, right inguinal. In the year 1854 her name appears again, age sixteen, hernia still right inguinal. In the following year there is a fresh entry, age seventeen, position of the hernia no longer inguinal, but femoral on the same, *i. e.* the right side. When examined by the writer there was no sign of inguinal hernia, but when the truss was removed a hernia escaped through the femoral ring. This patient's father and brother were also ruptured.

A young woman came at the age of twenty-six, with femoral hernia. She had been ruptured since the age of nine, and was still wearing a truss obtained from the society, and adjusted

by Mr. Taunton, for inguinal hernia of the same side. The femoral hernia appeared first after rising from her first parturition. Her father also had hernia.

In another case a young woman came at the age of nineteen, with inguinal hernia on the right side, which had existed since the age of sixteen. She had two sisters ruptured, both older than herself, who had become mothers before the protrusion appeared. In both the hernia was femoral. The father of the three was also ruptured.

These, and similar cases, add weight to the pathological theory of hernia. Rokitansky's unbiassed testimony corroborates it. When treating of the "anomalies and diseases of serous and synovial membranes," he says,[1] "Continuity of the sac of the peritoneum with the tunica vaginalis may be the result of partial development." And elsewhere he says,[2] "Arrest of development may occur as defective development of the mesentery in various points." Hence arrest of development (according to Rokitansky[3]) may result in the concurrence of an abbreviated mesentery with a patulent tunica vaginalis. Such a condition will explain the cases before alluded to, in which the tunica vaginalis remains open without protrusion. He elsewhere[4] speaks of hypertrophy of the peritoneum in the shape of unusual length of the duplicatures, *e. g.* omentum, mesenteries, &c., and of supernumerary folds and pouches, as a congenital anomaly. So that with the aid of Camper and Wrisberg's assertion about the early closure of the left tunica vaginalis, we have a ready explanation of the pathological origin of infantile hernia, and a clue to the interpretation of other forms. For instance, an infantile hernia due to hypertrophy of the peritoneum may be, and indeed generally is, outgrown in a few months by the increase of the capacity of the abdomen. So

[1] Sydenham Soc. transl., vol. viii, p. 17.
[2] Ibid., vol. ii, p. 12.
[3] Ibid., vol. iii, p. 17.
[4] Ibid., vol. ii, p. 12.

long as the child maintains his health and continues to grow, the hernia will not recur. But it is not at all uncommon for hernia to re-enter the tunica vaginalis about the time of puberty, or soon after, when the patient has reached (or nearly so) his full stature. If it be said, in interpretation of such a case, that the abdominal orifice was closed during growth, but opened again at puberty under the stimulus of sexual maturity, the pathological view is conceded. If, however, it be allowed that the orifice never closed, it must also be granted that the mesentery held the intestines away, and so hindered protrusion.

It cannot be doubted that there are many cases of hernia which, of necessity, owe their existence to a fault in the peritoneum. All congenital cases are of this order, and they constitute no small proportion of the entire list. If, then, it can be shown that all forms and kinds of the lesion, at whatever period of life, and under what conditions soever developed, are equally amenable to the same laws of inheritance, the inference will be reasonable if the conclusion be not inevitable, that all cases spring fundamentally from the same cause, viz., fault in the peritoneum.

Malgaigne[1] ascertained that nearly a third of the patients who came before him had other members of their family ruptured. The tables of the Truss Society show a somewhat larger proportion, slightly more than one third. This estimate is probably below the real proportion. Many of the patients who come to the society know nothing of their relations at all, or have so little intercourse with them as not to have learnt their condition in this particular, such as orphans, foundlings, emigrants, and foreigners. There are some who are ashamed of the malady, and conceal it from their relations, so that it is likely that nearly half the patients have ruptured relatives. But the tendency may be begotten in embryo—as a hybrid peculiarity might—without distinct inheritance. A young mother brought her

[1] 'Leçons Cliniques,' p. 41.

first-born child, a boy, to the Truss Society. She stated, in reply to questions put to her, that no other of her own or her husband's blood-relations than a nephew was ruptured. She and her sister had married two brothers; the first-born of each pair (both boys) were "born ruptured."

The tables of the Truss Society indicate a stronger inherited tendency in infancy and youth than in more advanced life. This may be partly accounted for. In childhood the patients are brought by one of their parents, or by some near relative from whom a history can be obtained. In later life the patients know less of their relatives.

The limits of this paper will not admit, nor does the argument require, the details of the inherited cases. It will suffice to say that, so far as has been made out, the laws of inheritance as laid down by cattle-breeders seem to hold. They say that[1] "peculiarities appearing in the males are transmitted either exclusively or in a much greater degree to males alone;" as also does another rule, viz., "that at whatever period of life a peculiarity first appears, it tends to appear in the offspring at a corresponding age, though sometimes earlier." Fathers, who themselves had hernia in infancy, but have outgrown the affection, beget children with congenital hernia, who also outgrow it. Brothers sometimes become ruptured about the same age, and before their father; and it is not uncommon for children to be brought with hernia in whom no family tendency can at the time be traced; but at the next visit, a year or so later, it will be said in answer to the customary inquiry that the father has become the subject of hernia in the interim.

These, and many more details necessarily omitted, indicate that hernia is hereditary, as much so as any other disease. The disposition not only attaches to families, but to races of men. It is said[2] to be rare in unmixed, dark-

[1] 'Darwin on Species,' p. 15.

[2] Knox, quoted by Teale, 'Ab. Hern.,' p. 38; and by Malgaigne, 'Leçons Cliniques,' p 28.

coloured, savage races, frequent in mulattoes. Lascars[1] are said to be rarely the subject of the malady. So far as the experience of the writer has gone, Irishmen are comparatively rarely ruptured. Very few have come to the society for relief during the past four years. Jews, however, seem to be very prone to the malady. Germans form a numerous class of patients of the society.

Freytag[2] and Blumenbach state that the Swiss are very liable to hernia. Malgaigne[3] and Knox state the Dutch to be strongly predisposed to it. Sir Astley Cooper[4] says the same of the Maltese.

Blumenbach[5] considers the gymnastic exercises to which the Swiss and Germans are addicted operate as a cause, while he considers the food of this people predisposes to the malady. They live, he says, almost entirely upon food derived from milk. Old cheese eaten with new cheese, instead of bread, is the mainstay of their living. Blumenbach also suggests that the milk diet of the Dutch is the principal reason of this great disposition to hernia. Soemmering suggests the indigestible nature of potatoes as a likely cause among some; but, as Mr. Lawrence[6] well says, "it seems strange that a single Irishman should escape the united operation of milk and potatoes. Since, however, ruptures are by no means particularly frequent in Ireland, where nineteen twentieths of the population subsist on these very articles, "either its natives must enjoy some peculiar exemption, or the learned writers quoted above must be mistaken." Nevertheless weakness, from whatever cause arising—whether from poverty and its consequent bad living, or from illness—is acknowledged by almost all writers to be a usual concomitant, if not necessary pre-

[1] Key and Hyslop; see Teale, as above.
[2] 'Lawrence on Rupt.,' 5th ed., p. 41, note.
[3] 'Leçons Cliniques,' p. 27.
[4] 'On Hernia,' 2nd ed., part i, p. 18.
[5] 'Lawrence on Rupt.,' 5th ed., p. 41, note.
[6] Ibid., p. 48, note.

cursor, of hernia. Sir Astley Cooper[1] lays stress on this. He says that weakness causes relaxation of the rings at the same time that it "operates in elongating the attachments of the viscera." He even considers[2] that inheritance acts through weakness of the fibrous structure of the rings; a view rightly disparaged by Malgaigne,[3] who considers it an influence persistent through life,[4] though manifested chiefly during the first third.[5] Nearly all the cases of recent hernia that have come under the notice of the writer have been associated with disturbed health, and often with asthenic bronchitis of a spasmodic sort.

Cases similar to those mentioned by Cooper,[6] Lawrence,[7] Richter, and others, in which persons habitually fat have become suddenly lean, and then have become the subjects of hernia, have also come under notice at the society, but very seldom. Fat persons are often ruptured when at their fullest bulk, as often, indeed, as at the time of their collapse. But if this sudden falling away be of itself a cause of hernia, there are few now who would accept Cooper's interpretation of its *modus operandi*. He says,[8] that "the fat which loaded the spermatic cord, and had extended the apertures to and from the abdomen, being suddenly absorbed, room is left for the viscera to supply its place." Now, as fat does not accumulate under pressure, it cannot, by loading the spermatic cord, dilate the rings. It is, however, stored up abundantly in the mesentery of fat persons, pushing the intestines forwards. This being suddenly absorbed would be likely to leave the intestines hanging lower than natural in the abdominal cavity, and so predispose to hernia. Cases sometimes occur in which all four inguino-crural openings and the umbilicus bulge with hernia at the same time, or in

[1] 'The Anat. and Surg. Treat. of Hernia,' 1804, ch. iv, p. 12.
[2] Ibid.
[3] Op. cit., p. 33. [4] Ibid., p. 41. [5] Ibid., 42.
[6] 'The Anat. and Surg. Treatm. of Hernia,' p. 12.
[7] 'On Rupt.,' 5th ed., p. 46.
[8] Op. cit., p. 12.

quick succession, as each hernia has been in turn controlled by a truss. In all these cases there has been a concomitance of great prostration, and general laxity of all the tissues.

Such cases are not manageable by mechanical appliances until the constitutional vigour has been in some measure restored. Until this has resulted the protrusion will pass the pads of the instrument, however accurately adjusted, like air-bubbles; but with improved health there comes also a more manageable condition of the hernia, so that men have been able to return to light work with safety.

Hence it is concluded that hernia is a disease, and not an accident; a pathological condition, and not merely a mechanical lesion; that although the proximate cause is mechanical, the remote, predisposing, real cause is pathological. The mechanical theory has been seen to break down, partly through disagreement of its advocates, partly through its failure to satisfy all the conditions of hernia; while the pathological theory, without interfering with the arguments in favour of the mechanical, supplies the complement.

It is frequently inherited, and subject to the rules that generally govern hereditary diseases; and when rapidly produced, its manifestation is generally associated with constitutional disturbance. It is therefore concluded to be itself a disease.

It has been shown, moreover, that the lesion is, in many cases, of necessity due to an abnormal condition of the peritoneum, parietal and mesenteric. Being subject almost equally to the same laws, regardless of age or occupation, it is argued that, in all forms of the malady, the fault is primarily attributable to the peritoneum in its entirety, and that protrusion is due partly to relaxation of the mesentery and partly to laxity of the parietal layer. It is also concluded that some of the conditions of hernia may exist alone without the protrusion of a hernial tumour. The mesentery may be relaxed or elongated alone, and give rise to what

Rokitansky calls "internal hernia,"[1] or may take the form, as in women, of prolapsus; and, lastly, it has been seen that apertures may in both sexes exist for years, perhaps for life, without protrusion, the parietal condition existing without the other.

If this be so it is clear that no operation, whatever its results, can rightly claim to be called a "radical cure," that aims solely at closing the aperture through which a hernia has passed. It is not the object of this paper to disparage the operation. Much value is claimed for it, and it is still on its trial; but inasmuch as its advocates adopt an attractive title, which assumes hernia to be a purely parietal lesion, it has been the writer's object to call in question its accuracy.

To have witnessed twenty-five failures argues of itself nothing against the principle of any operation, nor is it intended to adduce such experience now. If the principle be sound, a more perfected method, or a closer attention to detail, may produce more uniform success; but if the principle be wrong, these failures will, of course, enhance an antagonistic argument.

The object of the operation is to obtain obliteration of the neck of the sac of a hernia by artificially causing adhesion of its walls. If such a result were enough permanently to preclude the recurrence of hernia, a cure might be expected to occur oftener than it does as a consequence of the effects of strangulation. There are several cases on record,[2] and some must have been witnessed by many surgeons present, as they have by the writer, in which a fæcal fistula, the result by sloughing through the sac of a strangulated hernia, has healed and been followed, after a few months, by a fresh protrusion.

[1] Sydenham Soc. transl., vol. ii, p. 51.

[2] Dr. Heber Chase's edition of the 'Report of the Philadelphia Med. Soc.,' "On the Radical Cure of Hernia," 1837, p. 169; Jobert, 'Mal. Chir. des Can. Intest.,' tom. ii, p. 69; 'Lawrence on Rupt.,' 5th ed., p. 343; and others.

Suppuration about the sac of a hernia will cause perfect retention for some months. Sloughing of the subcutaneous cellular tissue in the region of the rings will similarly cause temporary retention, as, for instance, after extravasation of urine.

But even when a permanent cure results from the cicatrization of a sloughed sac and fæcal fistula, there has not heretofore been any evidence to show that the cicatrix made the obstacle to protrusion. Dupuytren's experience is otherwise[1]:—"We have had," he says, "the opportunity of examining, at the Hôtel Dieu, the bodies of several patients, many years after they had recovered by the mere powers of nature from artificial anus. We have found the intestine not attached to the parietes, as might have been expected, but loose in the cavity. A fibro-cellular cord, extending from the cicatrized portion of the abdominal parietes to the intestine, constituted the last vestige of the adhesions which had formerly united them." Scarpa gives similar evidence[2]:—"It is a certain fact," he says, "confirmed by a very great number of observations, that after the separation of a gangrene the two sound segments of the intestine retire gradually beyond the ring towards the cavity of the abdomen, notwithstanding the adhesions which they have contracted with the neck of the hernial sac." In these cases adhesions can have had nothing to do with retention of the hernia, because none existed, at least did not continue to exist. There were attachments, but so feeble as to be overcome by some internal traction, antagonistic to gravity in the erect posture, and to the expulsive force of the muscles. It would seem, then, that the sealing together of peritoneal surfaces by inflammation is unable of itself to oppose a sufficient barrier to the recurrence of protrusion, but that whenever a permanent retention follows such a process it must be due to some internal cause.

The only forces that can operate in detaching the intes-

[1] Quoted by Lawrence, op. cit., p. 369.

[2] Whishart's translation, p. 313.

tine from parietal adhesions are vermicular action, retraction of the mesentery, and the weight of the intestine when the body is horizontal. This last is slight, supplemental, and occasional, although, no doubt, it has an influence.

Retraction of the mesentery is probably the chief agent. Depuytren[1] acknowledges that it is through the medium of the mesentery that the intestine is released, but he seems to consider this membrane as purely a passive instrument, through which the motions of the spine effect the result. But even if it be so, there must still have been considerable retraction and shortening of the mesentery at the spot before it could be made useful in this way. It must first have recovered from the prolongation it suffered synchronously with the original formation of the hernia. Scarpa[2] rather attributes the recession of the intestine to "puckering of the cellular substance, which unites the neck of the hernial sac to the abdominal parietes within the ring," although he alludes to the possibility of its being "caused by the tonic and retractile action of the intestine itself and of the mesentery." It is likely that both these processes co-operate; but puckering of the parietal peritoneum can never produce the condition spoken of by Dupuytren, namely, the free mobility of the gut within the cavity of the abdomen.

Dissection has shown that the portion of mesentery attaching the protruded gut in the subjects of hernia is generally longer than the rest. This is acknowledged by all writers, but its cause has formed the subject of dispute. Before Scarpa wrote it was looked upon as one of the evidences of hernia, being the result of relaxation of the mesentery.

One old writer[3] went so far as to argue that "this relaxation of the mesentery is the effect of an extraordinary con-

[1] Quoted by Lawrence, op. cit., p. 368.

[2] Whishart's transl., p. 314.

[3] Benevoli; Scarpa, Whishart's transl., p. 43.

gestion of fluid, and principally of chyle, which runs through all the vessels of the mesentery, stagnates only in one division of this membranous support of the intestines, and relaxes only that portion of it corresponding with the hernia, while all the rest of the mesentery preserves its natural strength and tension." The extravagance of this view would seem to have driven Scarpa to an extremely opposite opinion, viz., that, by an excess of muscular power over resistance at the rings or weakest part of the walls, the intestine nearest this part is squeezed out, and the mesentery attaching it consequently stretched. The facts and arguments used in this paper point to the mean between these two views as the most nearly true. Relaxation of the whole mesentery will allow the small intestines to descend, and then the compression alluded to by Scarpa will be able to expel the portion least supported by the walls of the belly, and still further stretch the already relaxed mesentery. So long as the original debility continues, this stretching may go on; but returning health will check this process. That part of the membrane which has been supported may regain its wonted condition, while that which has been protruded and left without support will be set or established in its prolongation. When the illness is of short duration, and the protrusion obviated early by mechanical means, the whole of the relaxed peritoneum, mesenteric and parietal, will be likely to regain its pristine state, and result in a cure which will be justly entitled to the term "radical." Such a result is not uncommon in young persons. But even here the term must be limited, for the same predisposition which occasioned the opportunity for one protrusion will be likely, under the influence of constitutional derangement, to repeat that opportunity and give rise to a second.

Prolonged illness or defective mechanism will be followed by confirmed elongation of the mesentery, or its stretched portion, and pouching of the parietal peritoneum. This state of things will render a patient at all times liable to a

return of the hernia, though not necessarily the subject of continual protrusion.

Two patients volunteered the following accounts of their cases:—One, a man æt. 64, presented himself for a truss, with inguinal hernia on the right side. His story was that he was first ruptured at the age of nine years on the *left* side. He did not suffer from it, and had never worn a truss. It was always easily returnable. At the age of fifty he was thrown while riding, and pitched upon his head. From this time the hernia disappeared, nor has it ever appeared again on that (the left) side. For twelve years he remained without any hernia; but at the end of that time a protrusion descended through the right inguinal canal, and ultimately filled the scrotum. One of this man's brothers was also ruptured.

The other case was reported by the father when he came to the society for a truss. He said that his son was ruptured when a child, but had been perfectly cured ever since the age of sixteen, when he fell headlong into the water over a ship's side. He had been in the habit of wearing a truss before this accident, but had never needed one since, and several years had elapsed.

These were quite spontaneous statements. Two or three other accounts, very similar and quite as startling, have come under notice.

Many cases have come before the writer, at the Truss Society, in which supposed cures have resulted from the use of a truss on one side. The patients have remained sound for periods, varying from six months to as many or more years, and then the hernia has appeared on the other side.

The same thing has happened after the operation for strangulation,[1] the side operated on remaining sound, but hernia appearing after a year or two on the other side.

The readiest solution that can be offered is that the portion of intestine belonging to the elongated portion of mesen-

[1] 1862. Age 65.

tery had, in these cases, been shifted away from its old home into the region of its new protrusion.

The most "radical cure" is that which takes place in childhood. Here there is no need for a shrinking of the mesentery. The growth of the fleshy and bony boundaries of the cavity outstrips that of the superabundant peritoneum, and adjusts the relation of the chamber to its contents.

Analysis of the Patients with Inguinal or Femoral Hernia, during the

Ages.	Sex.	At the time of Examination.									
		Inguinal.				Femoral.				Inguinal and Femoral.	Gross Total.
		Right.	Left.	Double.	Total.	Right.	Left.	Double.	Total.		
0	Males...	103	46	35	184	—	—	—	—	—	184
	Females	5	—	—	5	—	—	—	—	—	5
1— 5	Males...	238	60	51	349	—	—	—	—	—	349
	Females	6	4	3	13	—	—	—	—	—	13
6—10	Males...	91	31	29	151	—	—	—	—	—	151
	Females	8	4	5	17	—	1	—	1	—	18
11—15	Males...	64	36	27	127	1	—	1	2	—	129
	Females	11	3	4	18	—	—	—	—	—	18
16—20	Males...	80	42	42	164	3	—	—	3	—	167
	Females	12	8	1	21	1	4	—	5	—	26
21—25	Males...	101	56	63	220	3	—	1	4	1	225
	Females	13	4	6	23	3	4	5	12	—	35
26—30	Males ..	116	69	139	324	4	1	—	5	3	329
	Females	15	17	10	42	18	9	8	35	—	77
31—35	Males...	155	72	162	389	2	4	1	7	5	401
	Females	17	17	8	42	19	14	10	43	—	85
36—40	Males...	123	113	207	443	8	3	5	16	3	462
	Females	14	12	10	36	16	15	9	40	1	77
41—45	Males...	170	122	209	501	4	3	5	12	5	518
	Females	15	13	14	42	36	8	16	60	2	104
46—50	Males...	151	87	254	492	8	7	9	24	5	521
	Females	15	18	13	46	25	15	24	64	2	112
51—55	Males...	124	74	225	423	5	5	8	18	13	454
	Females	6	12	9	27	15	11	15	41	2	70
56—60	Males...	110	62	203	375	8	3	9	20	11	406
	Females	10	10	6	26	19	9	15	43	—	69
61—65	Males...	104	51	191	346	8	2	6	16	14	376
	Females	5	5	7	17	19	9	7	35	1	53
66—70	Males...	72	39	121	232	4	4	7	15	7	254
	Females	8	3	5	16	4	11	8	23	1	40
71—95	Males...	65	31	114	210	3	2	3	8	5	223
	Females	1	4	5	10	8	3	3	14	1	25
TOTALS	Males...	1867	991	2072	4930	61	34	55	150	69	5149
	Females	161	134	106	401	183	113	120	416	10	827

who were relieved by the City of London Truss Society year 1863.

At time of first Discovery.									Family History.					
Inguinal.				Femoral.										
Right.	Left.	Double.	Total.	Right.	Left.	Double.	Total.	Gross Total.	Paternal.	Maternal.	Both.	Collateral.	Descends.	Total.
380	126	82	588	—	—	—	—	588	125	59	5	28	6	223
12	10	4	26	—	—	—	—	26	9	2	1	—	—	12
141	32	11	184	—	1	—	1	185	32	28	4	6	1	71
26	15	7	48	2	—	—	2	50	5	2	1	—	—	8
67	45	4	116	1	—	—	1	117	22	11	3	7	1	44
14	7	2	23	1	—	—	1	24	9	3	1	2	—	15
90	53	9	152	2	—	1	3	155	28	8	2	17	2	57
12	14	—	26	4	1	1	6	32	11	2	1	1	1	16
205	129	42	376	5	5	—	10	386	110	19	5	28	2	164
20	9	1	30	10	9	1	20	50	13	3	1	2	—	19
222	153	44	423	8	4	1	13	436	107	22	3	36	6	174
19	19	6	44	20	11	8	39	83	14	7	—	6	—	27
238	163	76	477	3	6	3	12	489	104	18	2	52	4	180
22	27	11	60	50	19	9	78	138	20	14	3	9	3	49
201	163	53	417	8	8	2	18	435	87	15	4	50	6	162
10	12	5	27	32	18	5	55	82	16	3	1	5	3	28
200	153	48	401	9	9	1	19	420	77	9	2	40	2	130
19	17	3	39	37	22	3	62	101	14	8	3	10	3	38
186	122	44	352	13	3	1	17	369	62	13	2	31	7	115
6	14	5	25	38	12	6	56	81	13	7	1	6	1	28
143	99	39	281	12	9	3	24	305	42	7	1	34	5	89
6	8	4	18	16	13	2	31	49	5	5	—	1	2	13
93	66	22	181	5	8	1	14	195	38	4	1	15	2	60
3	5	3	11	10	12	2	24	35	8	1	—	1	—	10
90	64	25	179	11	4	1	16	195	29	2	—	3	1	35
3	7	1	11	8	6	2	16	27	2	2	—	2	1	7
55	29	16	100	—	1	1	2	102	19	2	1	6	1	29
2	4	—	6	7	5	—	12	18	1	1	—	1	—	3
26	15	12	53	1	2	1	4	57	7	1	—	6	1	15
3	—	—	3	—	3	—	3	6	—	—	—	—	—	—
17	8	5	30	1	—	1	2	32	3	2	—	3	2	10
—	1	1	2	2	—	—	2	4	—	—	—	—	—	—
2354	1420	536	1310	79	60	17	156	4466	892	220	35	362	49	1558
177	169	53	399	237	131	39	407	806	141	60	12	46	14	273

REPORT OF THE COMMITTEE

APPOINTED BY THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY

TO INQUIRE INTO THE

USES AND THE PHYSIOLOGICAL, THERAPEUTICAL, AND TOXICAL EFFECTS OF CHLOROFORM,

AS WELL AS INTO THE BEST MODE OF ADMINISTERING IT, AND OF OBVIATING ANY ILL CONSEQUENCES RESULTING FROM ITS ADMINISTRATION.

Received June 14th—Read July 5th, 1864.

In laying their report before the Council of the Society, the Committee on Chloroform desire to state that they have made comparatively little reference to the medical portion of the subject. This is not due to their thinking the medical uses of chloroform of little importance, but to the fact that but few replies to their inquiries upon this subject have been received.

In view of the great extent of the questions submitted to their consideration, the committee directed their attention to such points as appeared to them of chief practical importance. Thus, their observations respecting the action of chloroform on the nervous system, and their remarks on some other points, are less full than would have been desirable, had the committee regarded such details as of equal importance with those specially elected for investiga-

tion, such as its influence on the action of the heart and on respiration.

The committee have chiefly confined their physiological report to observations which they have themselves made. Without overlooking or neglecting the labours of former investigators, they have endeavoured rather to furnish an accurate account of experiments which they have observed carefully and together, and to compare the results thus obtained and agreed upon, with the phenomena of cases in which death or peril of life has arisen from the inhalation of chloroform in the human subject.

How Chloroform arrests Animal Life.

In order to observe the manner in which animal life is destroyed by the inhalation of chloroform, a series of experiments (chiefly in dogs) were made, in which various proportions of chloroform vapour in atmospheric air were respired. The experiments with the more dilute forms of the vapour were conducted by means of Mr. Clover's apparatus, in consequence of the exactness with which the quantity of chloroform administered through it can be regulated. The effect of air impregnated with from 1 to 15 per cent. of the vapour was thus observed. When the results produced by chloroform in the strongest possible dose were under investigation, an inhaler was used in which the temperature of the chloroform was raised to about 150°, and its evaporation was thereby much accelerated. By this method the air inhaled was charged with at least 40 per cent. of the vapour.

In the first experiments the chloroform was administered, as in the human subject, either by a closely fitting inhaler or by a saturated cloth. As, however, in these experiments the breathing was sometimes arrested, apparently by spasm about the throat, it became necessary to compare the results obtained from natural breathing with those produced by administering the chloroform through an aperture in the trachea.

In the first instance attention was chiefly directed to the order of cessation of the pulse,[1] the respiration, and the action of the heart.

It was found that the duration of animal life was inversely proportionate to the strength of the agent; the more concentrated the vapour the shorter the duration, and the more diluted the chloroform the longer the continuance of life. Insensibility could be induced and maintained when the air inhaled contained no more than 1 or 2 per cent., and the inhalation might be continued for a very long time without apparent danger to life.

The *strongest doses of chloroform* inhaled through the mouth and nostrils caused the pulse and respiration to cease nearly simultaneously (in from 1 min. 20 sec. to 1 min. 45 sec.), whilst the heart's action continued for a short time subsequently (from 3 min. 10 sec. to 5 min. 30 sec.).

If, in place of respiring the chloroform by the natural channels, vapour of the same strength was inhaled through an aperture below the glottis, death ensued much more rapidly, and the heart, as a rule, ceased to beat several seconds before the final arrest of the respiratory movements. The average duration of the pulsations of the heart was 16 sec., whilst the respiration ceased at 32 sec. Owing to the rapid and tumultuous course, and the early termination of these experiments, the exact moment at which the pulse ceased to be perceptible could not be noted with accuracy.

With *moderate* or with *small doses* little difference was observed, whether the chloroform were inhaled above or below the glottis. Generally, but *not always*, it happened that the respiration ceased a few minutes (from 3 to 7) before the arrest of the action of the heart.

In the majority of cases the pulse stopped before the respiration, and in all instances the action of the heart could be distinguished for some time after the pulse had ceased to be felt.[2]

[1] In the femoral artery.

[2] Appendix A, Table 1.

Effect of Chloroform on the Heart.

It has been observed, in all instances of poisoning with the smaller doses of chloroform, that the pulse is imperceptible for some time before the heart cases to beat.

From this it is plain that the heart becomes enfeebled before its contractions finally cease. The extent of this failure of the force of the heart's action was marked by the hæmadynamometer.

The results of the experiments made with this instrument being remarkably uniform, the conclusions to which they point deserve attentive consideration. The instrument, having been set at zero, was connected with the femoral artery. The mercury at once rose, indicating the pressure of blood in the vessel. A double pulsation was then observed—a greater one, corresponding with the movements of respiration; and a lesser oscillation, timing with the pulsations in the arteries. The point at which the mercury stood after the hæmadynamometer had been connected with the artery having been noted, the first and immediate effect of the administration of chloroform was to cause the mercury to rise. This sudden elevation of the mercury was observed in nearly all instances, but was much more strongly marked in some cases than in others. Its extent could not be accurately measured, but it was sometimes as much as one and a half inch. The elevation was, however, of very short duration, seldom continuing for more than a fraction of a minute, and at the end of this period the mercury commenced to fall. The rise of the mercury (usually) corresponded to the period of struggling, and, no doubt, to a great extent, depended upon forcible expiratory efforts. A certain amount of elevation, however, was observed in cases in which there was but little struggling. This early elevation of the mercury in the hæmadynamometer must, therefore, on the whole, be considered to indicate that the immediate effect of small doses of chloroform on the heart is to stimulate its action.

After this transient rise of the mercury a gradual fall was noticed, and it sank lower and lower as the influence of the chloroform augmented. This falling of the mercury, however, as will be presently seen, was liable to certain interruptions. Thus, in Exp. XX, before chloroform was given, the mercury ranged from 9 to 14.[1] After the usual temporary use it had fallen in 3 min. to from 6 to 8·5. Then, after many variations, it gradually fell, till at 25 min. 45 sec. it ranged only from 2·4 to 2·6; and, at length, when all movement had ceased, the mercury stood at 2·1.[2]

Moreover, it was observed that the mercury did not subside steadily and uniformly from the commencement of the inhalation of the chloroform till the time at which the heart ceased to beat; but that there were both slight variations and also many periods at which the heart's contraction recovered its former force. If these revivals of the heart are observed in the tables (Exp. XX), they will be seen generally to follow the periods at which the respiratory movements had been diminished. In other words, it appears that if the respiration becomes slow and shallow, the force of the heart's action returns. The reason of this is obvious: by the failure of the respiration the introduction of the poison into the system is lessened, and the heart revives.

A still more striking circumstance was noticed when the force of the heart's action was fully under the influence of chloroform. If, at that moment, the respiration of fresh air was permitted, the mercury at once rose, and the heart in a short time recovered much of its force. Upon renewing the inhalation of the chloroform, the mercury promptly sank again. This experiment could be frequently repeated, and with each admission of fresh air there was revival, and with each repetition of the administration of chloroform there was depression of the action of the heart. (Exp. XXXIII.)

[1] Centimètres. [2] Appendix A, Table 4.

Movements of the Heart after the Rhythmic Contractions have ceased.

Upon carefully watching the state of the heart, it was ascertained that it not unfrequently retained some power of movement after the cessation of its regular action. In many instances these movements were prolonged for several minutes after the death of the animal; in others the heart appeared to have been at once paralysed; and, although it was exposed as quickly as possible after death, its movements had entirely ceased.

There are fourteen observations bearing upon this point. In six of these the heart was exposed in periods ranging from 3 min. to 6 min. 55 sec. after the cessation of the heart's regular contraction, and all movement was found to have ceased. Of these cases, the strongest form of chloroform had been employed in three, in two others 10 per cent., and in the sixth 5 per cent.

On opening the chest after death, in five other instances pulsation was found to have ceased, but rhythmic contractions recommenced after laying open the pericardium. These continued for some little time, their duration ranging from 4 min. to 15 min. 30 sec. In two of these cases, chloroform had been given in the strongest form, in two 5 per cent., and in the other $2\frac{1}{2}$. The instances in which the pulsations of the heart continued longest (12 min. 40 sec. and 15 min. 30 sec.) were the two in which the strongest form of chloroform had been administered.

In three other cases rhythmic contraction of portions only of the heart was observed when the chest was opened after death. In these instances the period during which the movement continued varied from 3 min. to 13 min. 15 sec. Of these cases two had inhaled 10 per cent., the third 5 per cent. Here, also, it was noted that the longest duration (8 min. and 13 min. 15 sec.) of the muscular contraction occurred in the cases in which a strong form (10 per cent.) had been employed.

From these results it seems fair to conclude—*first*, that in many instances all movement of the heart is arrested very soon after its regular action has ceased. *Secondly*, in a few cases imperfect contraction may continue for some minutes after the stoppage of the normal movements. Even in these cases the movements of the heart do not continue so long as they are observed to do when life is destroyed by asphyxia. *Thirdly*, in many instances the heart is so far amenable to the action of stimuli that exposure to the air occasions an imperfect renewal of its beat.

Moreover, the effect of chloroform upon the heart varies remarkably with the strength of the vapour employed. It does not appear from these results that strong chloroform causes a more permanent stoppage of the heart's action than the milder form of this agent. Doubtless it may be that, in cases of poisoning by a strong dose of chloroform, the amount which finds its way into the blood is actually smaller than if a weaker vapour had been inhaled through a longer period. The heart in the former case may stop quickly, and yet, to a certain extent, be capable of recovery; whereas in the latter case, although continuing to beat for a longer time, it may at length be so overpowered with the poison as to be unable, after once ceasing, to resume its pulsations.

Effect of Chloroform on Respiration.

When the concentrated vapour of chloroform was inhaled through the mouth its immediate effect was to arrest respiration. This result depended upon a spasm of the fauces and glottis (see page 332), which was induced by the direct action of the vapour on these parts. The arrest of respiration, however, lasted but a short time, frequently only a few seconds, and actual inhalation of the chloroform then commenced. When smaller doses (under 6 per cent.) were administered, or as soon as the first irritation of the fauces produced by a stronger dose had subsided, and

breathing was resumed, respiration was found to be much quicker than before the inhalation commenced. The inspiratory efforts were at first deep, but by degrees they became more and more shallow. With this loss of depth the respirations for awhile retained their unnatural frequency, but after a time they became less frequent than natural.

The depth of the respirations became less and less, and after the stage of perfect insensibility was reached the amount of air entering the chest was extremely small. If the inhalation was still persisted in the movement was at last completely arrested.

This arrest of the respiration is not necessarily final; on the contrary, it frequently happened, and more especially if the amount of vapour inhaled had been small, that after some twenty or forty seconds the respiration recommenced.

If the vapour was still allowed access to the air-passages the respiration again quickly ceased. The breathing might hereupon be renewed a second time; and even a third time these phenomena might be repeated before actual death ensued.

This natural effort at recovery from chloroform poisoning is by no means an exceptional circumstance; indeed, it appears to be the rule in all cases in which a small percentage of chloroform is used. Hence it follows that if respiration ceases during the inhalation of a small percentage of chloroform vapour the removal of the vapour will in many cases permit of a complete recovery.

The explanation of this recovery of the respiration appears to be the following.

The entrance of additional chloroform is virtually interrupted by the stoppage of the respiration, whilst that which is already in the blood is gradually dissipated; the influence of the agent thus sinks to a degree which is no longer incompatible with the performance of the act of respiration.

The effect of division of the Pneumogastric Nerves in Animals already under the influence of Chloroform, and the effects of Chloroform in Animals in which these nerves have been previously divided.

In order to ascertain the effect of inhaled chloroform on the heart, apart from the influence exerted upon that organ through the pneumogastric nerves, the following observations were made.

It is well known that if, in a healthy animal, one of the pneumogastric nerves be divided, very little immediate effect is produced. If both nerves be severed the number of the respirations is at once reduced by about one half, and the frequency of the heart's action is increased in an inverse ratio. Should the animal be young, death generally ensues quickly, not as a direct effect of the divisions of the nerves themselves, or as a consequence of the interruption of their influence upon the lungs and heart, but from suffocation, caused by the falling in of the parts paralysed, such, for example, as the larynx. In adults this suffocation does not take place, and the parts about the larynx being rigid, life may be prolonged for several days, or, indeed, indefinitely.

If, now, an animal is placed under the influence of chloroform before the nerves are divided, these phenomena became modified, and are even in some cases absent. The respiration became only slightly less frequent than before the division of the nerves, and sometimes there was no perceptible alteration of the number of respirations. The pulse, however, became extremely rapid, though even thus it failed to reach the rate observed in cases in which the animals had not taken chloroform. In like manner, if chloroform was inhaled after division of the pneumogastrics, the discomfort of the animal was manifestly relieved, the breathing became more frequent and easier, and the chloroform appeared to bring about greater toleration of the loss of the function of these nerves.

Action of Chloroform on the Glottis and Fauces.

During natural respiration it may be seen that the epiglottis is raised with each inspiration, and that the vocal cords are separated to nearly twice their previous distance from each other. It may also be observed that slight mechanical irritation of the epiglottis or of the cords produces no effect.

When dilute chloroform vapour (5 per cent. or less) was blown upon the fauces or cords, very little inconvenience ensued, and the animal continued to breathe in a natural manner.

If, however, air saturated with chloroform was employed, an instant and violent effort at deglutition was produced; with this effort the whole pharynx was seen to become contracted, the larynx advanced, and the epiglottis became hidden by the act of swallowing. This act was repeated many times (the use of the strong vapour being continued), but it gradually became less vigorous, and after about a minute it ceased, the animal by this time generally passing under the influence of the chloroform. The epiglottis then became fixed; it projected forwards, both during expiration and inspiration; and the vocal cords approximated at each expiration.

If the vapour was still administered the epiglottis was seen to move slightly with each inspiration, and at length, as the animal passed fully under the influence and stertorous breathing commenced, the epiglottis flapped backward and forward with each expiration and inspiration.

If, on first administering the strong vapour, the epiglottis was gently raised, so as to expose the glottis to view, no spasm or contraction of the glottis itself could be observed.

In order to discover how far these conditions depended upon the direct action of chloroform, the same agent was administered by the trachea, and the movements of the fauces and glottis were watched from above. It was found that there was generally a single effort at deglutition;

after that a tremulous movement of the soft palate and of the parts around; and then, as the animal passed more fully under the influence of the chloroform, the epiglottis fell.

If, after division of the pneumogastrics in the middle of the neck, chloroform in a strong form were administered by the mouth, the same efforts at swallowing were observed, but they were not so frequent or so perfect as in an animal with the nerves undivided.

How Ether arrests Animal Life.

In order to compare the effect of ether with that of chloroform, this agent was employed in a similar manner to that described in the previous observations.

The phenomena produced by ether in the strongest and in the more dilute form are not essentially different; by either of them animal life may be destroyed; and, as in the case of chloroform, the extinction of life is proportioned in rapidity to the concentration of the vapour.

It was found that there was a general similarity in the results of experiments with ether and of those with chloroform, but that ether was much the weaker agent of the two. Effects produced by ether in a strong and by chloroform in a dilute form were accordingly somewhat similar.

Whilst, however, this general similarity may be traced in the action of ether and of chloroform, there is an important contrast in their influence on the heart. Chloroform depresses the action of that organ, and frequently kills by inducing syncope. Ether, on the other hand, exerts but a very slight depressing influence on the force of the heart's action.

Hence death, when produced by ether, is almost invariably due to the failure of the respiratory movement, and the heart is generally found to continue its pulsations for some time after the respiration has ceased.

With the strongest form of ether death takes place more rapidly if the agent is given by the trachea than when it is

breathed naturally, and in nearly all cases the respiration ceases for some time before the heart stops.

The average time at which the pulse ceased to be felt when the inhalation was carried on through the trachea was 2 min. 43 sec.; the respiration at 1 min. 48 sec.; the heart at 3 min. 57 sec.

Ether of the same strength, given by the mouth, caused the respiration to cease at 3 min. 27 sec.; the heart at 4 min. 15 sec.

The only case (in the experiments performed) in which the heart ceased before the respiration was one in which the ether vapour, in its strongest proportion, had been administered by the muzzle; and in this case slight respiratory efforts continued for 1 min. 15 sec. after the heart had ceased to beat. In this exceptional instance the force of the heart's contraction was well kept up till within a few seconds of its final failure.

With the more diluted ether vapour (10 to 25 per cent.) animal life was generally destroyed if the inhalation were continued for a sufficiently lengthened period, and death ensued in from forty to sixty minutes.

Some animals seemed to possess great power of resisting the action of ether; and in one case 15 per cent. of it was administered to a dog for more than one hour without producing any indication of approaching death, and the animal eventually recovered.

When death did ensue the same sequence of phenomena was observed as when that event was brought about by the stronger vapour, *i. e.* the failure of respiration preceded that of the heart's action. The average interval between the cessation of the respiration and of the heart's movement being 2 min. 3 sec. When the moment could be discerned at which pulsation ceased in the arteries, it was found that the pulse was arrested some few seconds before the respiration.

Effect of Ether on the Heart.

The essential difference between the action of chloroform and ether is to be found in the effect produced upon the heart. The first operation of both agents is to stimulate the heart and to augment the force of its contractions; but after this chloroform depresses the cardiac action, whereas ether appears to exert but little influence upon the muscular movement of that organ.

The first or stimulating effect of ether is both less sudden and more sustained than that of chloroform, and for some time the heart goes on beating with more than its natural force. Sometimes, indeed, even after insensibility has been induced, the mercury in the hæmadynamometer stands higher than before the administration of the ether. This vapour may therefore be regarded in a certain degree as a stimulant to the force of the heart's action.

Moreover, during the insensibility, the pressure of blood in the vessels is well maintained up to the moment when death is imminent; and then with ether the mercury only falls after there has been manifest failure of the breathing, whereas with chloroform the mercury generally falls even during the proper performance of the respiratory function.

It is necessary to state that there is considerable difficulty in comparing the result produced by these two agents, as the stupor arising from chloroform is so much more profound than that induced by the weaker agent.

Effect of Ether on Respiration.

As with the stronger forms of chloroform vapour, so also with the stronger forms of ether vapour, administered by the mouth, there is a temporary arrest of respiration; but in the case of ether this is less marked.

With small per-centages of ether vapour there is no actual arrest of the breathing, although the inhalation of it in those qualities causes the number and the depth of

the respiratory efforts to be diminished. After a short time the respirations become slow and full; and next, while their frequency rises, the range of their movement is reduced. At a later period the respirations become more frequent and shallow; by degrees the external muscles of respiration cease to perform their office, and the air enters only with the movement of the diaphragm. After a time the diaphragm also is still, and the breathing is completely arrested.

As in the case of chloroform, this cessation of respiration may not be final; and, indeed, with the weaker forms of vapour it seldom is so. In the course of some seconds respiration recommences; and, if the etherization be continued, the same phenomena may be repeated before death actually occurs.

Post-mortem Appearances in the Animals poisoned with Chloroform.

As a general rule, all the cavities of the heart were found to contain more than the natural quantity of blood, and those on the right side were much more full than those on the left.

These points were especially noticed in eighteen cases, and in all of them the right cavities were more or less distended; while those of the left side were filled (but to a less degree than the right) in fifteen. In two of the remaining instances (in which 5 per cent. of chloroform had been inhaled) there was but little blood in the left cavities, and in the third and last they were nearly empty. Extreme distension of the left cavities with blood was met with in three instances (all 10 per cent.); but the right side of the heart, although filled in all the cases, was noted as being tensely filled in six instances.

The *blood* itself was generally liquid; but in several instances well formed and large, but not very firm, coagula were observed. In all these observations the animals were examined a few minutes only after death.

The colour of the blood on the two sides of the heart was

noted in thirteen cases. That on the right side was much darker than the blood on the left in seven instances; it was slightly darker on the right in two. In three there was no perceptible difference in the colour, whilst in the remaining one the blood was much darker on the left side than on the right.

Moreover, these variations in the colour of the blood did not appear to depend upon the amount of chloroform which had been used. In the single example in which the blood on the left side was the darker 40 per cent. had been employed. Of the three in which the colour on the two sides was the same 40 per cent. had been used in one, and in the others 5 per cent. Of the two cases in which the blood on the right side was slightly the darker, one was an example of the inhalation of 10 per cent., and the other of 40 per cent. In the remaining instances, in which the hue of the blood was much deeper on the right side, the strength of the chloroform vapour had varied from $2\frac{1}{2}$ to 40 per cent.

The prevailing colour of the blood was a brownish-red. Its hue, on the right side, was in all cases dark, and in some was very deep. On the left side it was, in most instances, of a brighter tint than that in the right chambers, but in some the colour was a deep purple.

Microscopical characters of the blood.—The blood was in six instances examined by the microscope immediately after death. The result of these examinations showed that the blood-corpuscles have a tendency to become crenate, and that they do not collect so much in rouleaux as blood from a healthy animal.

In two of these cases no difference was observed in the blood taken from the opposite sides of the heart. In three more the characters of crenation of the outline and of non-isolation of the blood-corpuscles were more marked in the blood from the right side of the heart; in one of these instances, however, the blood from the left side was perfectly natural in appearance, the corpuscles being well formed and bi-concave. In the sixth case the blood-corpuscles from

the left side were slightly crenate, but were natural in their characters on the right.

The last instance was one in which 40 per cent., the others in which $2\frac{1}{2}$ or 5 per cent., had been inhaled.

Lungs.—In some few instances the lungs contained more than the natural quantity of blood, and were consequently rather dark in colour; but in the majority of cases they were bright and florid.

Extravasations of blood had, in many instances, occurred. The amount of these hæmorrhages was very variable, there being in some cases only slight specks of ecchymosis beneath the pleura at the edge of the lung, whilst in others there were large and numerous patches of pulmonary apoplexy. Although this condition was often found where but small per-centages (5 per cent.) of chloroform had been inhaled, the most marked examples were those in which the vapour had been given in its strongest proportions.

It should be added that artificial respiration had been resorted to in a few of these cases; many of the most marked examples of pulmonary hæmorrhage, however, were those in which no attempts at resuscitation had been made.

Liver, spleen, and portal system.—Some congestion of the liver and spleen, and distension of the portal vessels, were almost always observed. The amount of it varied extremely, and did not appear to depend upon any condition of the experiments. It certainly bore no proportion to the amount of chloroform which had been employed.

Brain and its membranes.—The head was examined in six cases some hours after the death of the animal. The chest had not been opened. In all of these the vessels on the surface of the brain were found full of blood, whereas those in the interior of the cerebral substance contained no more blood than usual.

From these facts it is clear that, although there may, in

certain cases, be an impediment to the free circulation of the blood through the lungs, yet the appearances after death has been caused by chloroform are very different from those observed when life has been destroyed by asphyxia. In death from chloroform all the cavities are distended, and the cases are only exceptional in which the left side is empty. The rule, however, is alike in both—that the cavities of the right side contain more blood than those of the left.

It may be stated that after-death appearances in man have been recorded in but a small number of cases, and that no satisfactory conclusions can be drawn from the accounts thus given. The results obtained by examining animals immediately after death from chloroform offer the best post-mortem evidence which can be at present obtained.

Means of avoiding Accidents with Chloroform.

One hundred and twenty-three cases have been collected[1] in which death could be positively assigned to the inhalation of chloroform.

Even this large number is probably far short of the aggregate mortality which must have been due to its use in various parts of the world. Many of the deaths, moreover, happened during trivial operations, which, without chloroform, are not attended with risk to life. Added to these, there are cases still in which life is placed in imminent jeopardy during the administration of chloroform, although it is not actually lost.

Facts so important have led the committee to give their anxious attention to devise or adopt means for obviating such accidents.

Attention is therefore directed—1st, to the agent employed; 2ndly, to the method of administering it.

1. *Effect of the mixture of Chloroform and Ether.*

If a mixture composed of from 2 to 4 per cent. of chlo-

[1] Appendix B.

roform vapour and 98 or 96 per cent. of atmospheric air be inhaled, there is little or no risk to life.

In some cases it is indispensable to employ as much as $4\frac{1}{2}$ or even 5 per cent. of the vapour. But if a larger dose (one 10 per cent.) be inhaled, alarming symptoms are liable to supervene. At times, even with every care, and with the most exact dilution of the vapour, the state of insensibility may in a few moments pass into one of imminent death.

It is therefore extremely desirable to obtain an anæsthetic agent which shall be capable of producing the requisite insensibility, and yet is not so dangerous in its operation as chloroform.

Ether, to a certain extent, fulfils these conditions, but its odour is disagreeable, it is slow in its operation, and gives rise to greater excitement than chloroform. The committee therefore concur in the general opinion which in this country has led to the disuse of ether as an inconvenient anæsthetic.

In the absence of any known substance possessing the required qualities various mixtures of chloroform and ether have at different times been resorted to. It might be expected that a mixture of these bodies would combine most of the required properties, and be at once more active and compendious than ether and less energetic than chloroform.

The known differences in the actions of the two anæsthetics suggest that, in a mixture of them, the more dangerous properties of chloroform would be neutralized or reduced by dilution.

This might particularly be inferred from the influence which they respectively exert on the heart's action; the one depressing it almost from the first, the other sustaining or but little diminishing its force. These expectations would be further confirmed by the opposite effect which the two agents produce when mixed with the blood.[1]

[1] In a recent paper, read before the Royal Society, Dr. Harley has described the effects produced by the admixture of chloroform and ether with the blood. He states that in the first place chloroform

In accordance with these considerations, the committee conducted experiments with mixtures of the agents combined in the following proportions by measure:

Mixture A.—Alcohol, 1 part;[1]
Chloroform, 2 parts;
Ether, 3 parts.

Mixture B.—Chloroform, 1 part;
Ether, 4 parts.

Mixture C.—Chloroform, 1 part;
Ether, 2 parts.

The first of these mixtures (A) was proposed several years ago, and employed by Dr. Harley.

The second and third are mixtures which it is believed have been extensively used in America.

It was found that the physiological effects of the mixture B were very similar to that of simple ether; an animal might inhale it for forty or fifty minutes, even in a tolerably strong form (15 per cent.), without destroying life.

The mixture, however, was open to the same objections as ether itself, the chief of which was the slowness of its operation. The length of time necessary to produce anæsthesia with it was so great as practically to preclude its employment.

diminishes the power of the constituents of the blood to unite with oxygen and give off carbonic acid, whereas sulphuric ether neither diminishes the absorption of oxygen nor the exhalation of carbonic acid by blood.

In the second place, chloroform has not nearly so powerful an effect in destroying the red blood-corpuscles as ether; the latter rapidly dissolves the cell-walls and sets the contents free.

In the third place, ether has a much more energetic effect in causing the constituents of the blood to assume a crystalline form.

Lastly, ether prevents the blood from assuming an arterial tint when agitated with air, while chloroform does not prevent the occurrence of this normal change in colour.—'Proc. Roy. Soc.,' p. 159, 1864.

[1] The sp. gr. of the liquids used in making this mixture are—

Alcohol	838
Ether	1497
Chloroform	735

When a sufficient quantity of this mixture was given to destroy life, the respiration was observed to cease some time before the heart's action. The force of the cardiac beat, moreover, as indicated by the hæmadynamometer, was well maintained throughout the period of anæsthesia.

The mixtures A and C were very similar to each other in their action. This quite accorded with the fact that the proportion of chloroform was the same in both. The mode of their action, moreover, was intermediate between that of ether and that of chloroform.

It was found in the human subject, as well as in animals, that insensibility might be induced by means of them with sufficient rapidity; that is to say, in from four to eight minutes in animals, and in from ten to fifteen minutes in man.

And, further, it was ascertained, in animals, that inhalation of the vapour in a strong form might be continued for thirty or forty minutes without destroying life. Indeed, it was only upon employing a concentrated form of the vapour, and after prolonged endurance of its action, that death ensued.

In nearly all the experiments in which the animal was at length destroyed the respiration ceased some little time before the heart's action; and in nearly all, including those even in which a strong vapour had been employed, there were temporary suspensions of the respiration, followed by recovery, such as have been described as produced by the inhalation of diluted chloroform vapour.

These mixtures exercised a much less depressing effect upon the action of the heart than chloroform alone. In this respect, again, the mixtures appeared to combine the qualities both of ether and of chloroform; it being clear that, at the same degree of insensibility, the depression of the heart's action was less with either mixture (A or C) than with chloroform.

These considerations tend to establish the fact that a mixture of ether and chloroform (such as A or C) is as

effective as pure chloroform, and a safer agent when deep and prolonged anæsthesia is to be induced, while at the same time it is sufficiently rapid in its operation to be convenient for general use.

It is quite possible that at some future time an anæsthetic may be discovered which will fulfil the required conditions yet more perfectly than either of these mixtures. In the mean time the Committee suggests that both of them should be more extensively tried than they have hitherto been in this country. Of the two mixtures preference is, in the opinion of the committee, due to A, on account of the uniform blending of the ether and chloroform when combined with alcohol, and probably the more equable escape of the constituents in vapour.[1] The alcohol which it contains probably stimulates and sustains the action of the heart.[2]

2. *Mode of Administration.*

The several effects produced by the administration of chloroform, as well as of other anæsthetics, are tolerably uniform if the same strength of vapour be employed; and there is much reason to suppose that the irregularities attributed to it have been in a great measure due to the uncertain degree of its concentration. Experiments upon

[1] The mixtures A and C have been tried, at the request of the committee, in about seventy cases in the London hospitals, and the evidence of this limited experience tends to show that they may be given with safety and with complete effect, although they take a longer time than chloroform (ten to fifteen minutes) to procure anæsthesia.

[2] Ether is a more volatile fluid than chloroform, and in a mixture of the two the ether evaporates more quickly than the chloroform. The relative rates of evaporation of the two was observed by placing a known quantity on a cloth and exposing it to the air; it was then found that the per-centage lost was, after exposure, for

3 minutes, ether, 89 parts; chloroform, 75 parts;
15 „ „ 93 „ „ 85 „

The fact that the constituents of a mixture escape in somewhat unequal proportions is proved by observing the sp. gr. of mixture before and after exposure, and it was found in all cases that after exposure the sp. gr. was, to a certain extent, increased, proving ether had escaped before chloroform.

the lower animals, however, equally with observation on man, prove that there is but a narrow limit between that strength in which the vapour may be safely inhaled and that which is likely to produce alarming symptoms, if not death. But whether the hazard originates in natural or in accidental causes, the conclusion must be the same—that it is extremely desirable to adopt a method of administration by which the quantity of the vapour actually being inhaled may be graduated.

The results of the experiments which have been detailed show that it is as desirable to measure accurately the strength of the vapour as to weigh the dose of a medicinal agent administered by the mouth.

The only apparatus at present known to the committee which fulfils the necessary conditions is that contrived by Mr. Clover, which appears to afford the greatest, if not absolute, safety in the administration of anæsthetics. At the same time the apparatus is open to objections, the chief of which is that it is not very portable, and, requiring some amount of experience in its use, it must frequently happen that chloroform, or an anæsthetic, must be administered when it is not available.

In the absence of an apparatus by which the proportion of chloroform vapour can be accurately graduated, the plan of administering the anæsthetic on a handkerchief or lint appears to be the least open to objection. This method ensures a sufficient mixture of atmospheric air with the vapour; and, if the handkerchief be held at a proper distance (one and a half inches from the mouth), there is but little fear of the air becoming impregnated with a dangerous proportion of vapour.

Resuscitation.

In the investigation of this subject the committee directed their attention especially to the following points:—A. (1) The period within which resuscitation may generally be accomplished. (2) The latest period at which resuscitation is possible. In reference to this latter point,

it was especially the object of the committee to ascertain to what extent the capability of resuscitation was regulated by the state of the respiration and of the heart's action.

B. It was attempted to distinguish the difference in the capability of resuscitation exhibited by animals poisoned by large and by small doses of anæsthetics respectively; and (C) also the differences in the power of reviving animals destroyed by pure chloroform and those poisoned by a mixture of chloroform and ether.

D. The comparative value of (1) artificial respiration, (2) the artificial respiration of oxygen, (3) galvanism.

There are many difficulties in arriving at very precise conclusions upon the subject of resuscitation. In the first place, it is by no means certain that the animals in which attempts at resuscitation were made would not have recovered without the artificial means of restoration. This will be seen to follow from what has been said of the mode in which death was brought about by moderate doses of chloroform and other anæsthetics. The respiration in most instances ceased, or nearly ceased, some time before the respiratory movements were finally arrested. After these periods of apparent death the respiration was usually restored; and this kind of flickering between life and death might be repeated twice, or even oftener, before the actual death. Attempts at resuscitation in such cases, soon after the interruption of the respiration, would gain for the artificial means which were used the credit of the recovery, whereas that event would really have occurred had they not been employed. It is accordingly proper not to attach too much importance to those instances of presumed resuscitations which have followed soon after the arrest of the respiration. In cases, however, in which natural breathing had ceased for a period of sixty seconds recovery without some artificial means of resuscitation would have been extremely improbable. On the other hand, where attempts to resuscitate have been postponed till after the cessation of the heart's action, it is right to attribute the recovery to the means employed, as in these instances it is

extremely doubtful if the revival could have occurred without some artificial means of effecting it.

A further obstacle in the way of rightly estimating the experiments in resuscitation arises from the extreme difficulty of obtaining precisely parallel observations. It has been stated that chloroform, and other anæsthetics, in moderate doses, cause death by enfeebling and paralysing the action of the heart, and that in most instances the respiration is the first to cease. While, however, at the time of the final respiration the heart's action is invariably enfeebled, in some cases it continues regular, with a steady beat, and gives rise to distinct pulsations in the arteries of the limbs; in other instances it is feeble, irregular, even intermitting, and its pulsations are imperceptible in the arteries.

From this it will be seen that two animals may be poisoned at the same period, and with equal doses of chloroform, and yet their positions shall be very different as regards the prospect of recovery; the one is hopelessly overpowered by the poison, the other easily recoverable. The frequent occurrence of differences so great renders the study of this subject one of much difficulty, and suggests the need of great caution in estimating the results of experiments.

The following appear to be the positive results which may be deduced from the experiments undertaken by the committee:

The simple failure of respiration whilst the circulation remains good almost always betokens a recoverable condition; some such cases would doubtless revive spontaneously, and a still greater number with the aid of the usual means of resuscitation.

If, after respiration has ceased, the heart continues to beat with regularity and with sufficient force to cause perceptible pulsation in the arteries, recovery with the ordinary means of resuscitation is probable; but if the heart has either altogether ceased to beat or has become irregular, and there be no arterial pulsation, then restoration by any

means is doubtful; even in these cases, however, under certain conditions, it is not absolutely impossible.

The failure of the circulation to any considerable extent always involves extreme peril; yet recovery is sometimes possible, even when the heart has actually ceased beating.

In these instances it does not appear of decisive importance whether the respiration has continued up to the time of this cessation or not. After the heart has stopped, however, recovery is but just possible, and is by no means the usual result of attempts to resuscitate. That which appears chiefly to regulate the result is the condition of the heart prior to its final contraction. When it has been acting with irregularity and feebleness for some time, the eventual recovery is rare; but if its action have continued strong up to the moment of its cessation, recovery is probable. From this it follows that a recovery is more likely to occur in an animal quickly poisoned with a large dose, than in one in which the heart's action has been enfeebled by the long-continued inhalation of a small dose. The explanation of this difference has been referred to before.[1]

In the experiments with mixtures of chloroform and ether the same observations held good, as far as the cases of insensibility induced by the small doses of chloroform.

The comparative value of different Methods of Resuscitation.—Of the different means available for restoring animation, suspended under the influence of anæsthetics, there was but little difficulty in distinguishing artificial respiration, as both the most efficacious and the most easily applied. The cold douche or continuous stream of water on the head was so manifestly inferior to this method in its restorative powers, that only a few experiments were performed with it.

The action of electro-galvanism and electro-magnetism is very decided, and many recoveries were effected with them in circumstances as unfavorable as those in which artificial respiration proved successful. In aid of that most valuable operation, either of them may doubtless be of service; but

[1] Page 329.

the habitual resort to them in desperate cases would too often involve a fatal loss of time.

In several instances in which a needle inserted in the heart had ceased to indicate any movement of that organ, the application of an interrupted and weak current of electro-magnetism or electro-galvanism to the needle restored the cardiac pulsations; and in some cases, even without the aid of any other artificial means, the animals recovered. The committee, nevertheless, cannot but regard these restorative agencies as practically of secondary importance, both because the requisite apparatus for employing them can rarely be at hand, and, still more, because the results of their application are neither so regular nor so certain as that of artificial respiration.

The experiments on resuscitation were attended with opposite results, according as the animal had been poisoned by large or by small doses of chloroform. Those poisoned by a large dose were, as has been already shown, more easily recoverable than those killed by a small one.

Practically, however, it must be remembered that poisoning by a small dose is altogether an exceptional circumstance, and presents conditions which are amply guarded against in the human subject. An animal, under such circumstances, would have been on the verge of death for some time before the actual cessation of the heart's action. Upon the first appearance of such symptoms in the human subject the inhalation would be promptly discontinued. It is with a large dose, on the contrary, that the symptoms of approaching death come on suddenly and without warning.

The effects of the treatment with oxygen gas will be seen on reference to the table of experiments; although acting sufficiently well, it is an agent which does not admit of practical application.

Resuscitation in the Human Subject.—From experiments on animals, and also from a consideration of cases of accidents with chloroform in the human subject, the committee is

strongly of opinion that the first and most important means of resuscitation is artificial respiration. Certain other methods may prove of service in aid of that, as the principal one; but they are all objectionable, in so much as they delay the commencement of the artificial respiration.

It is of the most pressing importance that artificial respiration should be commenced the moment alarming symptoms exhibit themselves. The delay, even of a few seconds, will doubtless, in some cases, destroy the only chance of life.

Artificial respiration should be practised in the manner known as Dr. Silvester's method, and as recommended by the Committee on Suspended Animation. Those who are conversant with the use of the bellows, adapted to artificial respiration by Dr. Marcet, may effect a yet more perfect and deep artificial breathing; since by means of it a much larger quantity of air may be made to enter and to leave the lungs, and one chief object, that of eliminating the chloroform, may be speedily accomplished.

For the same reason, mouth to mouth insufflation is a most valuable method of resuscitation. By it several good recoveries have been effected, a large quantity of nearly pure air being blown into the chest at each insufflation. In all cases in which it is employed the nostrils should be closed and the larynx should be pressed against the spine, to prevent the escape of air down the œsophagus.

With reference to the employment of galvanism, it may be noticed that the most powerful effects were those produced when galvanism was applied to the neck; and little difference was observed whether the poles were laid on opposite sides of it or, one being placed on the front of the neck, the other touched the lower part of the chest.

The power of the agent was increased by connecting one of the poles of the galvanic coil with a needle inserted into the diaphragm. In several instances, in which even the heart had ceased to move, striking results were obtained by applying the galvanism directly to a needle in the heart, the

other pole being in contact with some exposed portion of the integument.

Galvanism requires to be used only in a very moderate intensity, and it is necessary to employ it in an interrupted current, and to leave frequent intervals of repose. Strong and continuous currents appear rather to exhaust than to restore muscular activity.

Physiological Conclusions.

The sequence of the phenomena produced by chloroform inhalation in animals is similar to that observed in man; and, if the same per-centage of the agent be administered, the results produced are nearly uniform.

Chloroform at first increases the force of the heart's action; this effect is slight and transient.

When complete anæsthesia is produced by chloroform, the heart in all cases acts with less than its natural force.

The strongest doses of chloroform vapour, when admitted freely into the lungs, destroy animal life by arresting the action of the heart.

By moderate doses of chloroform the heart's action is much weakened for some time before death ensues; respiration generally, but not invariably, ceases before the action of the heart, and death is due both to the failure of the heart's action and to that of the respiratory function.

The danger attending the use of chloroform increases with the degree of stupor it induces.

Apparent irregularities in the action of chloroform mainly depend on the varying strength of the vapours employed, on the quality of the chloroform, and on the constitution of the patient.

Ether.—The action of ether is similar in many respects to that of diluted chloroform.

The vapour of ether at first increases the force of the

heart's action, and this effect is both greater and of longer duration than that observed with chloroform.

The stimulation is followed by a depression of the force of the heart's action; but, at the same degree of insensibility, ether does not depress the action of the heart to the same extent as chloroform.

Ether destroys animal life partly by enfeebling the action of the heart, but chiefly by arresting the movements of respiration.

The energy with which chloroform acts, and the extent to which it depresses the force of the heart's action, render it necessary to exercise great caution in its administration, and also suggest the expediency of searching for other less objectionable anæsthetics.

The slow and uncertain action of ether renders this agent an inconvenient anæsthetic; and, though it is capable of producing the requisite insensibility, and is less dangerous in its operation than chloroform, the committee concur in the general opinion which, in this country, has led to the disuse of ether.

A mixture of ether and chloroform is as effective as pure chloroform, and a safer agent when deep and prolonged anæsthesia is to be induced; though slow in its action, it is sufficiently rapid in its operation to be convenient for general use.

A mixture composed of ether three parts, chloroform two parts, alcohol one part (by measure), is to be preferred, on account of the uniform blending of the ether and the chloroform when combined with alcohol, and the more equable escape of the constituents in vapour; and the committee suggest that it should be more extensively tried than has hitherto been the case in this country.

Fauces and glottis.—If concentrated chloroform vapour be suddenly administered by the mouth, a spasm of the fauces is induced, which lasts for some seconds; afterwards, when the animal has inspired, the phenomena of asphyxia are, for a time, associated with those of chloro-

form poisoning, and death is finally induced as by dilute chloroform.

If partial insensibility be first induced by weaker chloroform, no spasm of the fauces ensues upon the sudden administration of the concentrated form of the agent.

Resuscitation.—Artificial respiration is the most certain means of restoring life after poisoning with anæsthetics.

Resuscitation may generally be accomplished by artificial respiration, after natural respiration has ceased, provided the heart continue to act.

Resuscitation may *sometimes* be accomplished by artificial respiration even after the cessation of the heart's movements; but this result is exceptional.

Galvanism resuscitates within the same limits as artificial respiration, *i. e.* with tolerable certainty, in cases in which the respiration only has failed, and sometimes after all movement of the heart has ceased. It is, however, far less to be relied on than artificial respiration.

Animals quickly rendered insensible by a strong dose are more easily recovered than those which have been gradually narcotized, even by a small per-centage of the anæsthetic.

Rules relating to the Administration of Chloroform.

Chloroform should on no account be given carelessly, or by the inexperienced; and, when complete insensibility is desired, the attention of its administrator should be exclusively confined to the duty he has undertaken.

Under no circumstances is it desirable for a person to give chloroform to himself.

It is not advisable to give an anæsthetic after a long fast, or soon after a meal, the best time for its administration being three or four hours after food has been taken.

It the patient is much depressed there is no objection to his taking a small quantity of brandy, wine, or ammonia, before commencing the inhalation.

Provision for the free admission of air during the patient's narcotism is absolutely necessary.

The recumbent position of the patient is preferable; the prone position is inconvenient to the administrator, but entails no extra danger. In the erect or sitting posture there is danger from syncope. Sudden elevation or turning of the body should be avoided.

An apparatus is not essential to safety if due care be taken in giving the anæsthetic. Free admixture of air with the anæsthetic is of the first importance; and, guaranteeing this, any apparatus may be employed. If lint, or a handkerchief, or a napkin, is used, it should be folded as an open cone, or held an inch or an inch and a half from the face.

Chloroform should invariably be given slowly. Sudden increase of the strength of the anæsthetic is most dangerous. Three and a half per cent. is the average amount, and $4\frac{1}{2}$ per cent., with $95\frac{1}{2}$ of atmospheric air, is the maximum of the anæsthetic which can be required; given cautiously at first, the quantity within this limit should be slowly increased according to the necessities of the case, the administrator being guided more by its effect on the patient than by the amount exhibited.

The administrator should watch the respiration of his patient, and must keep one hand free for careful observation of the pulse.

The patient who appears likely to vomit whilst beginning to inhale the anæsthetic must be at once brought fully under its influence, and the tendency to sickness will then cease.

The occurrence, during the administration of an anæsthetic, of sudden pallor, or of sudden lividity of the patient's countenance, or sudden failure or flickering of the pulse, or feeble or shallow respirations, indicates danger, and necessitates immediate withdrawal of the anæsthetic until such symptoms have disappeared.

On the occurrence of these symptoms, and especially if they should become so urgent as to threaten death from failure of

respiration, of heart action, or of both together, the following rules of treatment are to be observed:—

Allow free access of fresh air, pull forward the tongue and clear the mouth and fauces, keep or place the patient recumbent, dash cold water on the face and chest, and aid the respiratory movements by rhythmical compression of the thorax.

In the more threatening cases commence instantly[1] with artificial respiration, whether the respiration has failed alone or the pulse and the respiration together.

Galvanism may be used in addition to artificial respiration, but the artificial respiration is on no account to be delayed or suspended in order that galvanism may be tried.

Few, if any, persons are insusceptible of the influence of chloroform, from two to ten minutes being required to induce anæsthesia. The time, however, varies with age, temperament, and habits.

The mixture of chloroform, ether, and alcohol, should be given in the same way as chloroform alone, care being taken, when lint or a handkerchief is used, to prevent the too free escape of the vapour.

Use of Chloroform in Surgical Operations.

With heart disease the anæsthetic may be given in any case which requires an operation, although when there is evidence of a fatty, weak, or dilated heart great caution is demanded. Valvular disease is of less importance.

In phthisis, when an operation is unavoidable, anæsthetics may be given with impunity.

For all operations upon the jaws and teeth, the lips, cheeks, and tongue, anæsthetics may be inhaled with ordinary safety. By care and good management the patient may be kept under their influence to the completion of the

[1] 'Med.-Chir. Trans.,' vol. xlv, p. 491.

operation. In these cases blood, as it escapes, if not voided by the mouth, passes into the pharynx. If any small quantity finds its way through the larynx, it is readily expelled by coughing. In operations upon the soft palate, fauces, pharynx, and posterior nares, if sudden or severe hæmorrhage is likely to occur, it is not advisable to induce deep insensibility. In cases requiring laryngotomy and tracheotomy anæsthetics may be employed with safety and advantage.

For operations upon the eye, involving the contents of the globe, the use of anæsthetics is open to objection, on account of the damage which the eye may sustain from muscular straining or vomiting. If employed, profound insensibility should be induced.

In operations for hernia, and in the application of the taxis, anæsthetics act most beneficially. For most operations about the anus profound anæsthesia is positively demanded.

In the condition of shock or of great depression, as after hæmorrhage, the careful administration of anæsthetics diminishes the risk of an operation.

In all cases other than those specially referred to it is sufficient to state, so far as a mere surgical operation is concerned, anæsthetics may invariably be administered.

The continuous vomiting occasionally induced by and following upon the inhalation of anæsthetics may be injurious by consequent exhaustion, as well as by mechanically disturbing the repair of a wound. With this reservation, they do not appear to interfere with the recovery of patients from surgical operations.

Statistics.—The results of 2586 capital operations performed before, and of 1847 performed since, the introduction of anæsthetics, collected from all authentic available sources,[1] show that anæsthetics have in no degree increased the rate of mortality.

[1] Appendix D.

Use of Chloroform in Obstetric Practice.

A.—In Natural Labour.

The careful administration of anæsthetics during labour is not attended with special danger, there being no well-authenticated instance of sudden death recorded, either in this country or abroad, so far as is known to the committee, where they have been given by a medical practitioner; but the occasional occurrence of unfavorable symptoms demands the exercise of caution during their employment.

An anæsthetic given so as to produce deep insensibility will, in many cases, suspend both uterine contractions and the auxiliary powers of parturition, and this may be turned to account in turning and in instrumental deliveries.

Administered in a moderate degree, and under proper regulation, it occasionally protracts labour by weakening the expulsive powers, but in a large proportion of cases it does not do so.

It has a decidedly beneficial effect in promoting dilatation of the maternal passages.

Its employment in natural labour does not predispose to puerperal convulsions, apoplexy, or other like complications, on the part of the mother.

If used injudiciously, it may increase the number of cases in which instruments must be ultimately employed, but no such result is likely to follow its judicious employment.

The balance of opinion is nearly equal as to whether it predisposes to imperfect contraction of the uterus after delivery, and thus leads to post-partum and secondary hæmorrhage.

As a rule, it has no such after-effects on the nervous or vascular systems of the mother as to retard her convalescence, or render her more liable to any of the forms of puerperal disease. Many physicians believe that it rather favours subsequent convalescence. A small minority holds a contrary opinion.

It has no tendency, from its after-effects, to interfere injuriously with the function of lactation.

With very rare exceptions, and those doubtful, it has no injurious influence on the child.

B.—In Abnormal Labour.

Anæsthetics may be employed with advantage in various obstetrical operations, as turning, forceps, craniotomy, and extraction of retained placenta, rendering the patient passive in the hands of the practitioner, favouring relaxation of rigid tissues, lessening the suffering of the patient, and favouring convalescence by reducing the effect of shock and exhaustion. In many cases of turning, deep anæsthesia offers the additional advantage of suspending uterine contraction, and thus greatly facilitates the necessary manipulations; and in instrumental delivery generally it may be remarked that, unless anæsthesia be properly induced, the administration of chloroform is likely to prove more hurtful than useful, by rendering the patient less manageable.

It is not, as a rule, however, advisable to give anæsthetics during obstetrical operations, if the patient is much enfeebled by hæmorrhage; and if so given, they ought to be accompanied by the use of stimulants.

Anæsthetics may be employed advantageously to check the paroxysms in puerperal convulsions; but in the majority of instances their use will not enable the practitioner to dispense with other aids, such as bleeding, the omission of which may be neither prudent nor proper.

C.—Rules relating to the Administration of Chloroform.

There are no reasons for giving preference to ether over chloroform, the latter being much more desirable in obstetrical practice generally, the only exceptions being those in which chloroform notably disagrees.

In addition to those given for the administration of anæsthetics in ordinary cases, it is generally desirable to

observe the following rules during their administration in labour, subject to modifications at the discretion of the practitioner.

In natural labour begin to give them generally at or after the termination of the first stage; but they may be given earlier if the first stage is unduly painful, or if the os uteri resists dilatation.

Give them only during the pains, and withdraw them in the intervals.

When the fœtal head bears on the perinæum give them more freely, to promote relaxation and relieve the increased pain.

Withdraw the anæsthetic immediately after the child is expelled.

If the patient is depressed, or the pains are sluggish during its administration, an occasional stimulant may be administered.

In cases where it seems to interfere with the progress of labour it may be necessary to suspend its use for a time and re-apply it after an interval, or even to withdraw it altogether.

In turning and instrumental deliveries deep anæsthesia must be induced, as in surgical operations, and the administration should then be entrusted to a competent person, whose sole duty should be to attend to it.

In midwifery a special inhaler for its administration is not generally necessary or desirable; a handkerchief or towel, so folded as to prevent blistering of the face and to allow free admixture of atmospheric air, being sufficient for the purpose.

D.—Use of Chloroform in Diseases of Women and Children.

In the treatment of diseases of women an anæsthetic may be employed to facilitate diagnosis in very sensitive patients, or where a complete examination cannot be made without inflicting much pain. In cases of spurious pregnancy and phantom tumours, by relaxing the abdominal parietes, it may assist in demonstrating their true character, and, acting

in the same way, it may help to define more accurately the character and relations of other abdominal and pelvic tumours, or to detect feigned disease.

As a therapeutic agent, the inhalation and external application of chloroform in the form of a liniment may be usefully employed to allay pain in some cases of severe dysmenorrhœa, neuralgia, and the like.

There is accumulated testimony in favour of chloroform inhalation proving serviceable in various spasmodic diseases of women and children, as hooping-cough complicated with convulsions, spasmodic croup, epileptic seizures, and some other forms of convulsions in children, hysterical convulsions, epilepsy, and various muscular contractions in women.

Appendix A.

SELECTED EXPERIMENTS (1).

Table *showing the order of cessation of the respiration and heart's action in dogs subjected to the inhalation of chloroform vapour by the mouth and nostrils.*

Strength of vapour.	Insensibility.	Pulse ceased.	Heart ceased.	Respiration ceased.	Remarks
Strongest, on towel.	m. s.	m. s.	m. s.	m. s.	
I.	...	...	5.20	3.30	
II.	...	...	5.30	5.0	
LXVIII.	2.15	...	4.30	3.35	
LXIX.	...	...	5.5	4.15	
LXXVII.	1.30	...	7.15	6.0	
LXXX.	2.0	...	14.55	14.30	
LXXXI.	1.50	...	8.0	6.45	
40 per cent.					
III.	0.45	...	3.30	1.20	
IV.	1.0	...	4.45	1.45	
XXII.	1.5	1.30	3.10	1.30	
10 per cent.					
VI.	1.45	...	7.0	6.15	
XXIII.	1.40	12.40	15.5	14.55	
7½ per cent.					
LXXXV.	2.0	...	9.45	9.15	
5 per cent.					
LXXII.	4.15	...	30.15	27.20	
2 per cent.					
XXIV.	8.45	...	...	...	Recovered. Chloroform removed after fifty-six minutes. 128 minims of chloroform inhaled.
1 per cent.					
XXVI.	4.15	...	...		Recovered.
XXVII.	...	...	...	...	Recovered. The inhalation discontinued after twenty minutes, the animal still partly sensible.

TABLE *showing the order of cessation of the respiration and of the heart's action in dogs subjected to the inhalation of chloroform vapour administered through an opening in the trachea.*

Strength of vapour.	Pulse ceased.	Heart ceased.	Respiration ceased.	Remarks.
40 per cent.	m. s.	m. s.	m. s.	
X.	0.20	0.20	0.35	
XI.	0.15	0.15	0.15	
XII.	...	0.14	0.45	
14 per cent.				
VII.	4.5	5.15	2,5	Artificial respiration on cessation of natural.
VIII.	2.0	...	...	Recovered. Allowed to respire fresh air.
IX.	4.30	7.15	6.15	
10 per cent.				
XIII.	1.15	11.45	4 30	Artificial respiration.
XIV.	0.50	3.0	3.0	
XV.	1.0	5.0	1.15	
5 per cent.				
XVI.	1.15	8.0	5.0	
XVII.	18.14	25.0	18.14	Inhalation interrupted.
XVIII.	18.0	21.0	17.45	
XIX.	5.15	...	...	Artificial respiration.
2½ per cent.				
XX.	26.40	26.40	27.30	

TABLE *showing the order of cessation of the respiration and heart's action in dogs subjected to the inhalation of the mixtures.*

Mixture. Strength.	Mode of administration.	Insensibility.	Pulse ceased.	Respiration ceased.	Heart's action ceased.	Remarks.
		m. s	m. s.	m. s.	m. s.	
57. A. Strongest	Trachea	...	...	2.10	2.20	
58. A. Strongest	Trachea	...	0.15	0.55	5.25	
59. A. 10 p. cent.	Muzzle	3.45	...	19.15	19.45	Hæmadynamometer
60. B. 15p.cent.*	Muzzle	8.15	...	45.55	52.20	Hæmadynamometer (*strength increased) experiments.
62. B. 15 p. cent.	Muzzle	5.0	...	...	...	Continued 51 m. Re-
63. B. Strongest	Muzzle	...	...	1·15	1.30	[covery.
84. C.						
85. C. Strong	Muzzle	...	...	18.30	20.30	
73. C. Strongest	Muzzle	2.45	...	14.10	15.45	
74. A. Strongest	Muzzle	1.35	...	21.15	21.20	
75. A. Strongest	Muzzle	9.15	...	...	53.45	[ment.
76. A. Strongest	Muzzle	2.15	...	...	...	Resuscitation experi-

SELECTED EXPERIMENTS (2).

TABLE *showing the effect of chloroform inhalation on the heart's action and on the respiration.*

Experiment XXIII.—10 per cent. (240 minims in 2400 inches of air), administered by the muzzle.

Time.	Pulse.	Respiration.	Movement.	Heart.	Remarks.
m. s.					
...	25	6	5—10	...	
0.0	...	...	...	...	Chloroform commenced.
0.15	...	1	...	...	
0.45	19	?	...	...	Struggling since the commencement; doubtful if any respiration.
1.0	...	5	...	...	
1.15	25	6	...	...	Expiratory cries.
1.30	...	10	...	...	
1.40	...	...	...	...	Insensible.
1.45	19	7	...	...	Still moaning.
2.0	...	5	...	...	Stertor.
2.15	Very feeble	...	3	...	
2.30	...	9	2	...	Stertor; pulse hardly perceptible.
2.45	...	...	...	...	Needle inserted in chest.
3.0	...	6	...	...	Very little air enters chest.
3.15	0	5	<1	...	
3.30	...	11	...	...	Respiratory efforts, with some stertor; no air enters.
4.0	...	5	...	...	
4.15	19	...	...	...	Pulse returning.
4.30	...	10	3	...	Stertor.
4.45	23	5	3	...	
5.0	...	5	4	...	
5.15	25	3	5	...	
5.30	26	5	5	...	Stertor still continues.
6.0	...	7	2	23	
6.15	29	7	1	23	
6.30	...	7	2	...	Stertor continues.
6.45	45	...	...	...	
7.0	...	7	...	...	
7.15	29	8	1	...	Pulse stronger.
7.30	...	5	...	...	
7.45	33	...	...	...	
8.0	...	14	...	41	
8.15	32	12	...	...	No air enters.
8.30	...	14	...	...	
9.0	...	12	...	...	Still no air enters.
9.15	25	...	...	...	
9.30	...	12	...	...	Ditto.
10.30	...	...	...	...	

Experiment XXIII (*continued*).

Time.	Pulse.	Respiration.	Movement.	Heart.	Remarks.
m. s.					
10.45	32	13	...	...	
11.30	...	19	..	...	
12.0	...	18	...	...	
12.15	...	11	...	...	
12.30	...	20	...	...	
12.40	0	...	...	...	Pulse has again ceased.
13.0	...	34	...	...	Respiration shallow, expiratory.
13.15	...	...	...	23	
13.30	...	12	...	...	
13.45	...	...	...	30	
14.0	...	15	...	30	Respiration noisy.
14.30	0	...	...	26	
14.55	...	...	...	...	Respiration ceased.
15.0	...	...	...	14	
15.5	...	...	...	..	Heart ceased.

SELECTED EXPERIMENTS (3).

TABLE *showing the effect of chloroform inhalation on the number of the pulse and on the number and depth of the respiratory movements.*

Experiment XVIII.—5 per cent., by the trachea. Before the chloroform was administered the pulse 20, respirations 3.

Time.	Pulse.	Respiration.	Movement.	Remarks.
m. s.				
0.0	...	...	...	Chloroform given.
0.30	21	2	10	
0.45	28	3	12	
1.0	27	3	...	
1.15	25	4	10	
1.30	27	7	4	
1.45	30	28	...	Respiration expiratory.
2.0	24	...	...	
2.15	...	13	...	Pulse intermitting. Needle inserted through heart.
3.0	...	5	...	
3.15	28	7	3	
4.0	29	9	4	
5.0	26	11	...	
7.0	36	13	...	
8.0	28	12	...	
9.0	29	11	...	
10.0	28	13	2	
11.0	22	7	...	
12.0	15	6	...	
12.15	25	6	1	
12.30	...	5	...	
12.45	...	5	2	

Experiment XVIII (*continued*).

Time.	Pulse.	Respiration.	Movement.	Remarks.
m. s.				
13.0	28	6	...	Respiration irregular.
13.15	27	5	...	
13.30	...	5	...	A pause between each respiration.
13.45	31	4	...	
14.0	...	4	1	
14.15	28	4	2	
14.45	...	3	1	
15.0	25	5	...	
15.15	31	7	...	
15.30	23	7	...	
15.45	31	6	3	
16.0	...	8	...	
17.30	24	11	...	
17.45	30	5	...	Respiration stopped immediately after this.
18.0	27	0	...	
18.15	0	0	...	No pulse; no movement of needle in heart.
18.30	0	0	...	Ditto.
22.0	15	...	...	After a fresh insertion of the needle through heart.
22.30	16	...	...	
23.0	0	0	...	No movement, and none produced by fresh insertion of needles.

TABLE *showing the effect of chloroform inhalation on the number and depth of the respiratory movements.*

Experiment VII.—In this the chloroform (14 per cent.) was administered by the trachea. Before commencing, the respirations were 14 (in 15 seconds), the movement 20.

Time.	Pulse.	Respiration.	Movement.	Remarks.
m. s.				
0.0	...	...	...	Chloroform administered.
0.35	...	...	...	Violent struggling.
1.5	...	22	30	
1.35	...	26	18	
2.5	...	...	0	Respiration ceased; heart pulsating feebly (observed by means of a pin).
3.35	...	...	...	Artificial respiration with chloroform into lungs.
4.5	24	4	14	Heart beating more strongly; no pulse in femoral artery.
5.15	...	...	...	Heart ceased acting. Artificial respiration with *air* was then employed, but there was no return of the heart's pulsation.

SELECTED EXPERIMENTS (4).

TABLE *showing the effect of chloroform inhalation on the number and force of the heart's action, and on the number and depth of the respiratory movements.*

Experiment XX.—2½ per cent. (60 minims in 2400 inches air), administered by the trachea. Before commencement, pulse 24, hæmadynamometer 9—14 (extreme), respirations 4, movement 8.

Time.	Pulse.	Hæmady-namometer.	Respira-tion.	Movement.	Remarks.
m. s.					
0.0	...	...	...	...	Chloroform administered.
0.15	21	...	4	10	
0.30	21	...	5	7	
0.45	26	...	3	20	
1.0	29	...	7	5	
1.15	31	11—13	9	7	
2.15	35	11—12	13	...	Respiration irregular.
2.45	34	...	34	...	Respiration expiratory.
3.15	33	6—8·5	35	...	
3.45	32	5—8	...	...	Respiration too rapid to count; shallow.
4.15	35	7—9	28	2	Respiration expulsive.
5.0	33	5—7·5	50	...	Respiratory movement hardly any.
5.30	33	6—8	40	<1	Very shallow.
6.15	33	7·5—9	43	<1	Ditto.
7.0	38	ditto	34	1—2	
7.30	29	ditto	26	1—2	
8.15	20	7—9	22	1	
9.15	29	7—8·5	18	3	Respiration rather deeper.
10.30	31	6·5—8·5	17	...	
11.15	29	6·5—8	15	<2	Action of heart irregular.
12.45	29	6·5—8·5	15	...	A pause between respirations.
14.0	30	6—8	15	...	
15.15	29	6—8	15	...	
17.30	15	4—6·5	12	3	
18.0	16	4—6	11	3	Pulsation very irregular.
18.45	11	4—5	9	2	
19.15	13	3—4·5	13	2	
19.45	12	3—4	15	1·5	
20.15	13	3—3·5	17	...	
20.45	12	2·5—3·5	15	2	
21.15	12	2·7—3·4	19	1	
22.10	11	2·8—3	13	...	Respiratory movement very slight.
23.0	...	2·8—3·2	...	...	Respiration almost ceased.
23.30	11	2·8—3·3	8	<1	Respiratory movement extremely slight.

Experiment XX (*continued*).

Time.	Pulse.	Hæmady-namometer.	Respira-tion.	Movement.	Remarks.
m. s.					
24.0	...	2·8—3·2	0	0	No certain respiratory move-ment.
24.45	10	2·7—3	0	0	
25.15	9	2·2—2·5	0	0	
25.45	9	2·4—2·6	0	0	
26.0	8	...	0	...	
26.30	5	...	4	...	Respiration again commenced.
26.40	...	2.1	...	...	Heart stopped.
27.10	0	ditto	9	7	
27.30	0	ditto	0	0	
27.45	0	ditto	0	0	Pin inserted through heart; no movement of it. No fur-ther sign of life.

SELECTED EXPERIMENTS (5).

TABLE *showing the effect of chloroform inhalation on the heart's action and respiration, and the results produced by interruption of the inhalation.*

Experiment XXXIII.—In this experiment chloroform of the strength 2½ per cent. was used (160 minims in 6400 inches). Before giving the chloroform the respirations were 8 in 15 seconds; the force of heart's action, as shown by hæmadynamometer, 11—14.

Time.	Pulse.	Hæmady-namometer.	Respira-tion.	Remarks.
m. s.				
0.0	...	...	...	Chloroform administered by muzzle.
0.50	...	11—20	15	Struggling.
1.5	...	13—16	16	
1.20	14	...	14	Whining and struggling.
1.50	...	11—15	18	Still struggling.
2.5	...	...	15	
2.20	...	...	17	
2.35	17	11—15	...	
2.50	...	11—14	16	
3.20	...	11·5—13	...	Conjunctiva not sensitive.
3.50	...	11—12	20	Insensible.
4.5	...	...	...	Both pneumogastrics divided.
4.20	...	11—12	17	(No struggling for breath.)
4.35	...	11—13	24	
4.50	...	11—12·5	19	Respiration deeper.
5.20	...	11—12	12	
5.50	...	...	13	Pupil half the size it was before the division.

Experiment XXXIII (*continued*).

Time.	Pulse.	Hæmadynamometer.	Respiration.	Remarks.
m. s.				
6.5	24	...	12	
6.50	...	...	12	
7.5	...	10·75—11·75	...	
7.20	...	...	12	
7.35	...	...	...	Chloroform removed.
7.50	...	10·5—11	...	
8.5	...	10·5—11·5	7	Respiration deep.
8.35	...	11—12	8	
8.50	25	—13	...	
9.35	38	12—12·5	7	
9.50	...	...	6	
10.5	...	12—13·5	6	
10.50	45	12·5—13	8	No struggling for breath till present time.
11.35	...	...	...	Trachea opened.
11.50	...	—16	10	
12.20	...	14—16	16	
12.35	...	...	...	No struggling for breath.
12.50	...	15—15·5	...	
13.20	...	...	7	
13.35	50	...	7	
13.50	...	...	...	Sensibility returning; pupil smaller.
14.20	63	14—14·5	...	
14.35	...	...	6	
15.20	...	...	...	Chloroform again administered.
16.5	...	13—13·5	17	
16.20	...	...	15	
16.35	...	13—13·5	11	
16.50	...	12·5—13	...	
17.5	...	...	30	
17.20	36	9·5—10	36	
17.35	...	...	44	Conjunctiva insensible.
17.50	...	9—9·5	...	
18.5	...	...	...	Chloroform withdrawn.
18.20	...	10—10·5	...	
18.35	...	11—11·5	...	
18.50	...	12—12·5	13	Respiration deeper.
19.5	...	12—13	...	
19.20	...	...	13	
19.35	40	13·5—14	13	
19.50	...	14—14·5	20	Conjunctiva still insensible.
20.35	...	...	6	
20.50	...	...	...	Struggling; returning sensibility.
21.5	...	13—13·5	9	
21.20	...	...	9	
21.50	...	...	...	Chloroform again given.
22.20	41	...	11	
22.35	...	9—9·5	14	
22.50	...	...	11	
23.5	...	8—9	15	

Experiment XXXIII (*continued*).

Time.	Pulse.	Hæmady-namometer.	Respira-tion.	Remarks.
m. s.				
23.20	...	...	19	
23.35	39	8—8·5	39	
24.5	...	9·5—10	26	
24.20	...	...	34	
24.50	...	...	31	Insensible,
25.5	...	...	...	Chloroform removed.
25.35	...	9·5—10	...	
25.50	...	11·5—12	28	
26.5	...	11·5—13	22	Chloroform again given.
26.20	36	...	27	
26.35	28	12·5—13	20	
27.5	29	...	...	
27.20	...	...	15	Chloroform removed.
28.20	...	...	8	
29.50	...	...	...	Chloroform again given.
31.35	...	8—8·5	...	Fully under the chloroform.
33.50	...	...	...	Chloroform poured in trachea.
34.20	0	5—5·5	0	No pulsation in artery; no respiration for the last fifteen seconds.
34.50	...	—4·5	0	Mercury does not move.
35.35	...	...	7	Has again made seven respirations.
35.50	...	...	4	After four more efforts respiration ceased.

TABLE *showing the effect of the inhalation of ether vapour on the force of the heart's action.*

Experiment LIII. — 14 per cent. given by the muzzle to a small dog. Hæmadynamometer connected with femoral artery.

Time.	Pulse.	Hæmady-namometer.	Heart's action.	Respira-tion.	Remarks.
m. s.					
0.0	...	...	...	...	Ether given.
7.0	...	...	...	...	Some struggling and whining.
13.50	...	...	...	...	Cornea insensible.
21.0	...	...	...	...	Hæmadynamometer connected with femoral.
22.0	...	10—14	...	...	Animal perfectly quiet.
26.45	48	9—12	...	8	
29.45	45	9—12	...	13	
30.45	44	9—12	...	12	
35.45	. .	9—12	...	12	
37.45	...	...	...	...	Needle inserted in heart.
38.15	...	10·5—12·5	...	...	

Experiment LIII (*continued*).

Time.	Pulse.	Hæmady-namometer.	Heart's action.	Respira-tion.	Remarks.
m. s.					
42.0	...	...	...	...	A second hæmadynamometer connected with other femoral. Readings from the two alike.
43.0	35	11—13·5	...	8	
44.15	38	10—11	...	7	
44.45	...	...	...	0	Respiration stopped.
45.15	20	4·5—5·5	...	1	Respiration again commenced.
45.30	...	..	...	2	
45.45	...	4—6	...	5	
46.15	...	...	...	5	
46.30	26	5—8·5	...	5	
46.45	...	...	...	5	
47.0	...	...	...	5	
47.15	...	...	...	4	
47.30	38	...	...	4	
49.15	43	8—10	...	6	
51.45	17	...	...	2	
52.0	0	1½	...	4	Pulse ceased; no movement in hæmadynamometer; but pulsation (slight) continued in needle in heart.
52.15	0	...	...	4	
52.45	0	...	...	0	
53.0	0	...	7	0	
53.15	0	...	7	0	
53.30	0	...	3	0	
53.45	0	...	2	0	
54.0	0	...	2	0	
54.15	0	...	1	0	
54.30	...	...	...	...	Heart's movement quite ceased.

The direct action of the Vapour of Chloroform and of Ether on the post-mortem movements of the heart in Frogs.

In order to observe the effect produced by the action of the vapour of anæsthetics upon the cardiac movements, the hearts of several frogs were removed from the thorax, and, being denuded of pericardium, were exposed to the vapour of chloroform or of ether. The duration of the pulsations was noted, and the results thus obtained were compared with those observed in hearts removed in like manner

and simply suspended in moist air. The observations were as follows:

		a.m.
Exp. 1.—Heart removed from thorax and suspended in moist air. Contractions 36	at	9.58
Contractions 20 in the minute	„	10.30
Heart stopped	„	11.05
Exp. 2.—Heart suspended in air. 22 Contractions 56	„	11.15
Contractions 22 in the minute	„	11.40
Heart stopped	„	1.50
Exp. 1.—Heart suspended in vapour of ether. Contractions 28	„	10.22
Contractions 24 in the minute	„	10.25
Heart stopped	„	10.31 (9 minutes.)
Exp. 2.—Heart suspended in vapour of ether. Contractions 44	„	11.05
Contractions 28 in the minute	„	11.15
Heart stopped	„	11.20 (15 minutes.)
Exp. 1.—Heart suspended in vapour of chloroform. Contractions 34	„	10.21
Contractions 20 in the minute	„	10.25
Heart stopped	„	10.27 (6 minutes.)
Exp. 2.—Heart suspended in vapour of chloroform. Contractions 36	„	10.54½
Contractions 20 in the minute	„	11.3
Heart stopped	„	11.5 (10½ minutes)

Thus, both chloroform and ether appear to have a direct action on the heart, destroying its contractile power.

Appendix B.

ACCIDENTS WITH CHLOROFORM.

Fatal cases of Chloroform Inhalation.

The cases in which death took place during the inhalation of chloroform, and in which the fatal result was fairly attributable to this agent, are arranged in Table A. The number of cases in this table is 109 (72 males, 37 females).

The first 59 correspond to those collected by the late Dr. Snow. To these, however, have been added the cases numbered 7, 34, 35, 47, and 48, which had not been published when Dr. Snow's table was prepared. Moreover, the cases numbered 8, 29, and 46, although not admitted by him into his table, have also been added, since there seems no doubt that the death in each of them was due to the employment of chloroform. In two of these latter cases there existed fatty degeneration of the heart, which may doubtless have contributed to produce the fatal result, but which did not alone cause it. In the third case death was attributed to mental emotion. This, again, should be looked upon as a subordinate, not as the principal, occasion of death.

This collection of 109 cases cannot be regarded as comprising all the deaths which have taken place from the use of chloroform, since there is good reason to believe that many deaths have happened (especially out of England) which have never been made public. Dr. Snow, in addition to the fifty cases collected by him, alluded to six other fatal cases with which he had become acquainted. One, if not more, of these have since then been published; it has been added to the table now produced.

In table B are included nine cases in which death may have taken place from the inhalation of chloroform; but the fact cannot be regarded as sure, owing either to the imperfect reports or to the agent having been secretly used for the purpose of suicide.

If, however, these nine cases be admitted, together with the five cases of Dr. Snow, and the two classes be then added to those in Table A, the total (123) will represent the number of recorded cases in which death may fairly be attributed to the inhalation of chloroform.

It would be possible yet further to augment this number, by accepting as deaths produced by chloroform certain other instances in which the cause of the fatal result was either imperfectly authenticated or was erroneously alleged to be due to the inhalation of that agent. In all the cases included in the foregoing tables death took place either during or

immediately after the inhalation. Others have been published in which the death was attributed to chloroform, although it did not occur until some hours, or even days, after the inhalation. In nearly all these instances there were other causes (such as the ordinary effects of the operation performed), which might equally be charged with the death of the patient. That the chloroform contributed to produce the fatal result in these instances may be true; yet it is clear that they cannot with justice be included in Table A with the unquestioned cases of fatal chloroform inhalation. There is, indeed, some reason to believe that chloroform may combine with other causes to occasion death at some little time after its inhalation, but on this subject there is little satisfactory evidence. Thus, in Table C are arranged four cases in which death took place some time after the inhalation, in each of which there were other conditions capable of producing death independently of chloroform.

Age.—The ages in the fatal cases are as follow:

Under 5 years	0
From 5 to 15 years	9
„ 15 to 30 „	30
„ 30 to 45 „	32
„ 45 to 60 „	12
Over 60 years	2
Not stated	24
	109

Operations for which the Chloroform was administered in the Fatal Cases.

Amputations	16
Dislocations	5
Removal of tumours	9
Examination of injuries	3
Operation on male genito-urinary organs	12
„ on anus, rectum, &c.	7
„ on the uterine organs	1
„ on the eye	4
For hernia	1
Castration	4
For necrosis, excision of bones, &c.	3

Excision of joints	2
Forcible straightening of joints	3
For application of escharotics	6
Plastic operations	6
Ligature of arteries	1
Sinus in thigh	1
Impaction of fæces	1
For removal of teeth	12
Removal of toe-nail	5
For relief of neuralgia	2
For delirium tremens	2
For maniacal excitement	1
Not stated	2
Total	109

Mode of Inhalation.

On handkerchief, towel, or lint	55
Lint with sponge	5
On sponge	7
With the ether-inhaler	2
Snow's inhaler	5
An inhaler	21
Not stated	14
	109

Period of Inhalation at which Death occurred.

Under 1 minute	10
From 1 to 3 minutes	8
„ 3 to 5 „	10
„ 5 to 15 „	23
Over 15 minutes	4
Not stated	54
	109

The time was in nearly all cases arrived at by estimation, and was not noted by the watch. The results, therefore, above stated can only be taken with much allowance. The general conclusion, however, from the 55 cases in which the period of death is mentioned, is that the

fatal result in 51 cases happened within the first fifteen minutes.

Stage of the Anæsthesia at which Death occurred.

Commencing to inhale	10
Stage of excitement	16
Incomplete anæsthesia	24
Fully under influence	38
Ditto, operation complete	14
Not stated	7
	109

Or—

Before full effect of chloroform	50
During " "	52
Not stated	7
	109

Mode of Death assigned.

Syncope	56
Syncope during stage of excitement	6
Died suddenly	6
Died in a fit	10
Pulse and respiration ceased together	9
Failure of respiration (pulse not noted)	6
Failure of respiration (pulse remaining)	2
Not stated	14
	109

The mode in which death took place in the human subject corresponds with that observed in the lower animals. In ten cases death happened before the anæsthetic effect of the chloroform had been produced; and in these cases there is reason to believe that the death resulted from the large per-centage of chloroform vapour with which the air was charged. Although thus highly concentrated, the total quantity of the vapour consumed was in some of these cases remarkably small.

Then, again, many of the deaths may be attributed to the sudden inhalation of a dense chloroform vapour when the patient was already partly under the influence of this agent. This was doubtless the case in those instances in which syncope occurred during excitement.

From the very large proportion in which deaths by syncope, and not by failure of the respiration, predominate, the conclusion arises that overdose of the vapour had really occasioned the fatal result. The importance of this conclusion will at once appear from its necessary corollary, that, if efficient means of graduating the per-centage of the chloroform vapour had been adopted, the liability to death in many instances would have been diminished.

Yet it must not be supposed that in all the cases of death by syncope a per-centage of the chloroform vapour had been administered which was inevitably dangerous. In the experiments on animals some died by syncope whilst inhaling much diluted vapour, and in every case the force of the heart's action was much reduced for some time before death. If, as usually happened in the experiments on animals with the diluted chloroform vapour, the respiration gradually failed whilst the heart continued to beat with appreciable force, this constituted a sufficient warning of approaching death, and upon the withdrawal of the vapour recovery at once ensued. Few, if any, deaths have taken place in this manner in the human subject.

If, on the other hand (as sometimes, but rarely, happened), there was sudden failure of the heart's action, the breathing still continuing, death supervened, practically, without warning. This mode of death, which was exceptional in the experiments on animals, is the most frequent in the human subject. And this might, indeed, be expected, since, whilst the experiments on animals were purposely carried on to their conclusion in order that all the symptoms of chloroform poisoning might be observed, man would be guarded by precautions and attention which would commonly secure him from all dangers to life but those which could not be recognised or averted during their approach,

as from the early inhalation of a large quantity of the anæsthetic by sudden and deep inspiration.

It is noted that attempts were made to resuscitate the patient by means of artificial respiration in 49 cases. This, in 23 cases, produced no effect, but in 26 there were efforts at natural respiration. These efforts soon ceased, and the patients died.

The period at which the attempt to resuscitate was made is exactly noted only in a few cases; it may, however, be assumed that artificial respiration was very promptly commenced in the great majority of cases.

TABLE A.—*Fatal Cases of Chloroform.*

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
1	Jan. 28, 1848. Reported by Dr. Meggiston, at Winlaton. Hannah Greener, æt. 15	Removal of toe-nail. Death during operation	Handkerchief. One dram used. Incomplete anæsthesia. Two minutes	Became blanched; spluttered as in epilepsy; made no rally.
2	Feb. 23, 1848. 'Med. Gazette.' Cincinnati. Martha Simmons, æt. 35	Extraction of teeth. Nearly completed	Dr. Morton's ether inhaler. Large quantity used. Fully under influence. Death, three minutes	Arms became rigid. Pulse ceased after being feeble; respiration ceased about the same time. Became livid.
3	March, 1848. Mr. Warren. 'Med. Gazette.' Boston. Patrick Coyle	For fistula	Towel or handkerchief Half a dram used. Anæsthesia incomplete (?). Death, one minute	Showed slight symptoms of feeling pain. The pulse, which was full and natural, sank.
4	May, 1848. Boulogne. Madlle. Stork	Opening a sinus in thigh. Death probably took place before the operation was commenced	Handkerchief. Three drams used. Anæsthesia incomplete Death, one minute	Put up her hand, said "I choke." The face immediately became pale; breathing embarrassed. She foamed at mouth.
5	—— 1848. 'Med. Gazette.' Hyderabad. Female, young	Amputation of middle finger. Death probably took place before the operation was commenced	Handkerchief. One dram used. Anæsthesia incomplete. Probably one or two minutes	She coughed a little; then gave a few convulsive movements.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred and time.	Mode of death.
6	May, 1848. 'Med. Gazette.' Hôtel Dieu, Lyons. Charles Des Noyers, æt. 22	Cautery to wrist. Operation commenced	An apparatus. Not stated. Five minutes	Not stated.
7	June 25, 1848. M. Robert, 'Bull. Acad. de Médecine.' Hosp. Beaujon, Paris. Male, æt. 24	Amputation at the hip, for bullet wound. During operation	An apparatus. Death on reapplication of chloroform; when sensibility was returning. Period not stated	Breathing became stertorous, and then very feeble; countenance livid; eyes turned upwards; the pulse had ceased. Artificial respiration appeared to revive him, and the pulse was again felt; he relapsed, however, and died.
8	June 28, 1848. Mr. Robinson, 'Med. Gazette.' London. Mr. Walter Badger, æt. 23	Extraction of teeth. Not commenced	An ether apparatus. Incomplete anæsthesia. Three or four minutes	Hands fell down, and head fell on chest. Artificial respiration, but no sign of recovery.
9	Dec., 1848. 'Med. Gazette.' Govan. Male youth	Operation on great toe. Not commenced	Not stated. Almost instantly	Not stated.
10	Jan. 19, 1849. Dr. Warren, 'Med. Gazette.' New York. John Griffith, æt. 31	For phimosis. Had inhaled chloroform before. Completed	Napkin. Three drams used. Complete anæsthesia. Ten minutes	Face and neck became livid; the eyes turned upward; pulse imperceptible at the wrist; and whole body relaxed. After two or three gasps, he ceased to breathe.
11	Jan. 24, 1849. 'L'Union Méd.' Hôtel Dieu, Lyons. J. Kenier (male), æt. 17	Amputation of finger. Not commenced	Gauze. Two drams. During stage of excitement. Six minutes	Pulse ceased to beat; countenance altered. Action of heart had ceased, and the sounds could not be heard. Respiration still continued, but became irregular, weak, and slow; and at length ceased completely, in the space of half a minute. Attempts made to restore respiration, and in two minutes again commenced; but pulse did not return.
12	Feb. 20, 1849. 'Lancet.' Westminster. Samuel Bennett, mason, æt. 36	Amputation of a toe. Completed	Handkerchief. About half an ounce. Complete anæsthesia. Time not stated	At close of operation no blood escaped when the pressure was removed from the arteries; the patient was, in fact, dying, and in a short time expired. A few inspirations were noticed after the pulse had ceased at the wrist.

13	Aug. 23, 1849. 'Lancet.' Langres, France. Madame Labrune, age not stated	Extraction of tooth. Not commenced	Handkerchief. Quantity not stated. Incomplete insensibility. Time not stated	As she did not become insensible, more chloroform was placed on handkerchief. She drew one deep inspiration; countenance immediately became pallid; dilatation of pupils, convulsive rolling of eyes; no pulse could be felt.
14	Oct. 10, 1849. 'Lond. Med. Gaz.' St. Thomas's Hospital. John Shorter, æt. 48	Removal of toe-nail. Completed	An inhaler. About half a dram. Complete anæsthesia. Five minutes	Continued insensible after operation; face becoming dark; pulse small, quick; respiration laborious. After struggling for a minute, became still. Respiration continued a few seconds after cessation of the pulse. Artificial respiration produced no effect.
15	—— 1849. 'Journal of Prov. Med. and Surg. Assoc.' Shrewsbury. Female, age not stated	Excision of the eyeball. During operation	Sponge. "A small dose." Probably fully under. Time not stated	She died suddenly.
16	Jan., 1850. 'Lond. Med. Gazette.' Female, æt. 20	Extraction of tooth. Operation seems to have been attempted several times at intervals unsuccessfully. Died when inhaling chloroform for sixth time	Sponge and napkin. Commencing to inhale	Died suddenly, stretching herself out, and frothing at the mouth at the moment of death, the countenance at the same time becoming livid.
17	Jan. 29, 1850. 'Edin. Month. Journal.' Kingston, Jamaica. William Bryan, age not stated	Operation not stated. Probably not commenced	Sponge. About one dram. Stage of excitement	Made a stertorous inspiration, and after some seconds made another inspiration; and this occurred several times, until at length respiration ceased entirely. (No report of pulse).
18	Feb., 1850. Board of Ordnance. Mauritius. An artilleryman, æt. 24	Amputation of finger. Probably completed	Handkerchief. One dram	After chloroform was discontinued face turned pale, pulse and breathing ceased.
19	—— 1850. 'Hygiea.' Stockholm. Male, æt. 30	Operation on testicle. Not commenced.	Cotton and folded towel. About two and a half drams. Five minutes	Some struggling on breathing fresh chloroform. Towel removed, but, patient not being insensible, again applied. After a few inspirations pulse ceased, face turned pale, breathing slow, and gradually ceased.
20	March, 1850. Dr. Snow, from Dr. Adams. Glasgow. Male, æt. 7 or 8	Sounding for stone. During this operation	Lint. Insensible moaning	Countenance livid, eye vacant. Heart pulsation ceased. Made one deep gasp, and was to all appearance dead. Artificial respiration; no result.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
21	June, 1850. 'Med. Gaz.' Guy's Hospital. Police-constable, æt. 34	Amputation of portion of hand. During operation	Inhaler, then napkin	During operation hæmorrhage suddenly ceased, and he expired.
22	Sept. 20, 1850. Dr. Snow, from Dr. Adams. Cavan. Ireland. James Jones. æt. 24	Amputation below knee for scrofulous disease of ankle. Not commenced	Lint and towel, with sponge. About one and a half drams. About one minute	Slight convulsive movement of eyelid, froth at mouth, and he was dead.
23	April, 1851. Dr. Snow (newspaper report ?). Stepney Workhouse. John Holden, age not stated	An operation on penis	Not stated. One dram	Died suddenly.
24	June 10, 1851. 'L'Union Médicale.' Strasburg. Madame Simon, æt. 36	Extraction of teeth. After extraction of some	Handkerchief. Less than one minute	Died suddenly.
25	July 8, 1851. 'Med. Times.' Seaman's Hospital, Greenwich. A mulatto, æt. 45	Removal of testicle. During operation	Linen cloth. About one and a half dram. Fully under influence. More than seven minutes	Hæmorrhage from artery suddenly ceased, and the pulse at the same moment. Respiration ceased almost instantaneously with heart's action, but one or two sigh-like inspirations followed. With artificial respiration there were several natural efforts at respiration.
26	Oct., 1851. 'Med. Times.' Chipping Norton. Elizabeth Hollis, æt. 37	Removal of impacted fæces. (Cancer of uterus.) Completed (?).	Not stated, probably handkerchief. Ten and a half drams. Fully under influence. Eight or nine minutes	On completing operation the surgeon found that she had ceased to breathe.
27	March 17, 1852. 'Med. Times.' St. Bartholomew's Hosp. Thomas Haward, æt. 23	Ligature of arteries, aneurism in leg by anastomosis. Operation commenced	An apparatus. Five to ten minutes	Pulse suddenly ceased; chloroform removed, but in a few seconds the patient had ceased to breathe. With artificial respiration the act of respiration was performed several times, and the circulation was observed to be returning, but he quickly relapsed and died.

28	June 27, 1852. 'Gaz. Médicale.' Ulm. Madame W—, æt. 32	Extraction of tooth. Not commenced	Sponge and handkerchief. Twenty-five drops. Only a few inspirations	On being asked a question, answered in a thick and trembling voice. At the same time stretched out her limbs; the face became bluish; the eyes haggard; the head and arms fell; she was dead.
29	Sept. 15, 1852. Dr. Snow, 'Anæsthetics.' London. Male, æt. 73	Lithotrity. During operation	Snow's inhaler. Fully under. Fresh chloroform given, as signs of returning consciousness appeared. Time not stated	Appeared to hold his breath; the pulse had then ceased, the heart's sounds were still heard feebly. He made several further inspirations, and then ceased to breathe. Artificial respiration produced no result.
30	—— 1852. 'Med. Times and Gaz.' Melbourne, Australia. Mr. J. Atkinson, age not stated	Fistula-in-ano. Not commenced	Handkerchief. About one dram. Not fully under. Not more than a minute	On applying fresh chloroform he spluttered at mouth. The chloroform was removed, but he suddenly expired.
31	Aug. 10, 1852. 'Monthly Journ. of Medicine.' Melrose, Scotland. A cattle-dealer, age not stated	Application of potassa fusa to ulcers. During operation	Inhaler nor quantity stated. Fully under influence. A few minutes	When nearly completing operation a sort of catch in breathing on looking at him; the mouth and eyes open, and turned upwards; the breathing irregular; face pale, and the pupils dilated. Artificial respiration no effect. "In a few minutes the man died."
32	Dec. 24, 1852. 'Lancet.' Manchester Infirmary. Henry Hollingsworth, age not stated	Removal of malignant tumour of the thigh. During operation	An inhaler. Quantity not stated. Fully under. Chloroform acted slowly	Breathing became slow; he seemed to be sinking fast; he gave one strong gasp, and then died.
33	March 19, 1853. 'Lancet.' University College Hospital. Caroline Baker, æt. 28	Application of nitric acid to sloughing ulcers. Not commenced	On lint. Quantity not stated. Stage of excitement. Time not stated	A partial relaxation of the limbs took place; she became insensible and pulseless.
34	May, 1853. M. Triquet, 'La Patrie.' Paris. Madame Breton	Tumour of the face. During the operation	Compress. Stage of excitement	Had cough; made a deep inspiration, then a convulsive movement of the face and hand; the pulse and heart had ceased.
35	—— 1853. M. Valleix, 'Gazette des Hôpitaux.' Paris. Male	For hæmorrhoids, in a patient suffering from aneurism of the aorta. Not commenced	Not stated. Fully under. Under five minutes	The respiration became embarrassed; there was trismus; then the movements of the heart ceased. With insufflation, and then with electricity, he breathed irregularly for twenty minutes. There was, however, no evidence of restored action of the heart.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
36	May, 1853. 'Med. Times and Gazette.' Hôtel Dieu, Orleans. A soldier, æt. 25	Removal of tumour near lip. Commenced	On sponge. Five grammes. Fully under. About four minutes	Patient became pale, respiration suspended, sank into a state of complete collapse. Artificial respiration used, but produced no effect.
37	Sept. 28, 1853. 'Monthly Journal.' Edinburgh Infirmary. John Mitchell, æt. 43	Perineal section. Not commenced	Handkerchief. About an ounce. Fully under. Time not stated	Had a slight convulsion; rallied, and continued chloroform; pulse became weak, and ceased. Breathing did not cease before pulse. Artificial respiration restored a few natural respirations, but this did not continue.
38	Oct. 5, 1853. 'Med. Times and Gazette.' University Hospital. Female, æt. 40	Strangulated hernia. Not commenced	Lint. Less than two drams. Stage of struggling. Five or six minutes	Struggled much; commenced to breathe with loud, rough stertor; the pulse was gone; gave several inspirations, then ceased. With artificial respiration and galvanism gasped about three times. After this no further signs of life were exhibited.
39	Oct. 20, 1853. 'Medical Times and Gazette.' St. Bartholomew's. Ann Smith, æt. 22	Application of actual cautery to cancroid sore. Not commenced	An inhaler. About 2½ drams. Fully under. About five minutes	Pulse became extremely weak and fluttering; countenance dusky; respiration at long intervals, with slight catching efforts. All efforts at respiration ceased about two minutes after first indication of failure; the pulse, however, as a very feeble flutter, was felt occasionally for at least two minutes later.
40	Nov. 16, 1853. 'Edin. Monthly Journ.' Vienna. Male, æt. 19	Extension of anchyloid knee. Not commenced	Inhaler. Quantity not stated. Probably not under influence. Less than a minute	Pulse became frequent and undulating; trismus occurred; the respiration became irregular, face livid; foamed at mouth. He only once made a feeble attempt at respiration.
41	—— 1853. 'Med. Times and Gazette.' Neustedt. Female, æt. 13	Lipoma on back; removal. Commenced	Not stated. One dram. Fully under. Time not stated	Fell suddenly forwards on her chest. Attempts made to restore her; but, in the course of a few minutes, it became evident that she was dead.
42	Jan. 21, 1854. 'Assoc. Med. Journ.' Bristol Infirmary. Jane Morgan, æt. 59	An attempt to reduce dislocation at shoulder. Not commenced	Sponge. Two drams. Stertorous breathing. About six minutes	Breathing became stertorous, and immediately afterwards the pulse, which had hitherto continued pretty firm, became suddenly imperceptible, the respiration ceasing at same time. There was, with galvanism, "some convulsive efforts of respiratory muscles, but no further sign of life."

43	—— 1854. 'L'Union Médicale.' Hôp. St. Antoine, Paris. Female. æt. 40	Removal of uterine polypus. Almost completed	Lint. Quantity not stated. Fully under. Time not stated	Pulse ceased to beat; face was extremely pale; a slow respiration still continued, but soon ceased.
44	—— 1854. 'Assoc. Med Journ.' Sheffield. Mrs. Harrup, æt. 45	Removal of cancer of breast. Not commenced	Neither form of inhaler nor quantity stated. Fully under. More than forty minutes	After inhaling with little effect for forty minutes the chloroform took effect, but the countenance changed and the pulse ceased; after a few short laboured inspirations life became extinct.
45	May, 1854. 'Med. Times.' Lock Hospital. A tailor, æt. 18	Operation for phimosis. Not commenced	Inhaler (not covering nostrils). Two drams. Incomplete insensibility. About six minutes	Pulse suddenly failed, became imperceptible; countenance assumed a pale, leaden hue. With stimulus of cold water, after three or four minutes, pulse again felt, and spontaneous respiration renewed. This improvement continued for ten minutes; then pulse and respiration ceased together. Artificial respiration produced no effect.
46	May 11, 1854. Mr. Potter, 'Med. Times and Gaz.' St. George's Hospital. Female, æt. 37	Removal of tumour of breast. Not commenced	Snow's inhaler. Rather more than one dram. Commencing to inhale. One minute and a half	Breathing suddenly ceased; became deadly pale, no pulse could be felt; there were then two sighing efforts at respiration Artificial respiration commenced within one minute, without effect.
47	June 1, 1854. M. Nistri, 'Gazette Hebd.' Pisa. Male, age not stated	Reduction of dislocation of the hip. Reduction effected	An apparatus (which mixed air with the chloroform). Complete anæsthesia. A few minutes	Pulse ceased; became pale; the respiration continued, but slow and irregular. Friction, ammonia, artificial respiration; the arms were raised, and some pulsations were again felt. He appeared to answer questions, but then fell back again into a state of syncope.
48	—— 1854. MM. Vattelle, Perrin, and Lallemand. Hôtel Dieu, Lyons. Male, æt. 13	For cleft palate. During the operation	A compress, held two centimètres in front of mouth. Fully under	The pulse ceased; there was a strange expression of face. With artificial respiration and cautery there were two or three efforts at respiration.
49	July 13, 1854. 'Medical Times.' Middlesex Hospital. Labourer, æt. 65	Amputation in the thigh. Not commenced	Snow's inhaler. Three drams. Stage of excitement. Fourteen minutes	Pulse, which was full and steady, gave a few rapid, irregular beats, and then ceased; respiration ceased simultaneously; face became pallid and death-like. Artificial respiration was followed by a slight effort at inspiration.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
50	Oct. 11, 1854. 'Medical Times.' University Hospital. Shoemaker, æt. 39	Introduction of catheter. During operation	Lint. Quantity not stated. Fully under. Time not stated	Began to snore; then a long interval; the breathing became feeble, and was ceasing; artificial respiration then used, and after a few minutes it recovered, but again ceased. The pulse had continued to beat some little time after these symptoms and after cessation of respiration.
51	Dec. 5, 1854. 'Med. Times and Gazette.' Guy's Hospital. Female, æt. 56	Amputation of leg. Not commenced	Lint, in oil silk. Two drams. Fully under. About three minutes	Pulse suddenly ceased in femoral; then several respirations, which ceased, but were renewed slightly with artificial respiration.
52	April 10, 1855. 'Med. Times and Gaz.' Ophthalmic Hospital. Male, æt. 40	Excision of eyeball. Not commenced	Dr. Snow's inhaler. Stage of excitement	Respiration became feeble and sighing. With artificial respiration there was occasional breathing, but this presently ceased. Pulse, when felt for after spasm subsided, was absent.
53	Sept. 8, 1855. Dr. Snow. London. Female, æt. 29	For relief of facial neuralgia	An inhaler. Half dram. Not fully under	Having inhaled twenty minims, she begged for more; began to inhale eagerly; gave a sudden start, as if taken in some kind of fit. No further sign of life.
54	—— 1855. 'Edinb. Med. Journ.' Edinburgh. Female, æt. 36	Not stated. (Tooth extracted?) Not commenced	Handkerchief. About a dram and a half. Quite conscious. About a minute	Spoke, and said, "I am not over yet;" and immediately, while yet speaking, she gave a convulsive start, and, with a stertorous inspiration and with the eyes and mouth open, sank to the floor. Artificial respiration was employed, and after a short time there were a few spontaneous inspirations, and it is said the pulse could be perceived at the wrist.
55	Oct., 1856. 'Med. Times and Gaz.' St. Thomas's Hospital. A sailor, æt. 30	Removal of necrosed bone from finger. Not commenced	Sponge in lint. Not fully under. Time not stated	Raised hands, and trembled; kept spitting at the lint; appeared as if about to vomit. Suddenly he was violently convulsed, as if in an epileptic fit. The chloroform was at once discontinued, and he was laid in a semi-horizontal posture. The convulsion lasted only a few seconds; he began to breathe with effort, and to gasp irregularly. His pulse was almost imperceptible, and intermittent. With artificial respiration he rallied, and breathed without assistance. In a few seconds he relapsed, and could not be recovered.

56	Feb. 28, 1856. 'Med. Times and Gaz.' London. Male, æt. 9	Excision of scapula. Not commenced	First an inhaler; then lint. Under influence. A few minutes	After making one long, deep inspiration (from lint), appeared to pass into a deep sleep. A few seconds later pulse began to beat very quickly, then ceased for two or three seconds, then beat rapidly several times and ceased. He continued to breathe for at least a minute longer. Respiration was ceasing, but continued, with cold water, &c., for two or three minutes. Brandy poured into mouth passed into œsophagus, but no effort to swallow. Artificial respiration produced some efforts at inspiration; but he did not rally.
57	April 5, 1857. 'Lancet.' Liverpool Infirmary. A labourer, æt. 35	Amputation of the thigh. Not commenced	Lint. Not a large quantity. Fully under. A few minutes (ten minutes from the time on table till death, but had inhaled in the ward three or four minutes before)	On raising eyelid it remained retracted; eyes slightly turned up. No pulsation in temporal or wrist. The respirations had almost ceased. With cold water, &c., respiration became better; in about two minutes a pulse was felt at the wrist. This continued, with good respiration, two or three minutes. The pulse then failed, and then the respirations. Artificial respiration produced no effect.
58	Aug. 7, 1857. 'Med. Times and Gaz.' Female, æt. 17	Application of nitric acid to sores. Completed	Snow's inhaler. One dram. Fully under. A few minutes	The operation being completed, moved as though recovering, and was left. A few minutes later, being noticed to be pale, was found pulseless and dead. Artificial respiration produced no effect.
59	March 13, 1858. 'Brit. Med. Journal,' p. 207. Reported by Mr. Prichard. Bristol Infirmary. Wm. Howell, æt. 49	Excision of elbow-joint. Operation not commenced	A hollow sponge. About one dram of chloroform used. Not under influence. About two minutes	Gasping respiration followed by "slow convulsive movement of limbs." The pupils became dilated and the pulse stopped.
60	April 17, 1858. 'Med. Times,' p. 415. Toronto. Male, age not stated	Extraction of teeth	No particulars	"Died suddenly."
61	May 1, 1858. 'Med. Times,' p. 457. Bonn. Male, æt. 23 (intemperate)	Plastic operation on forehead. Not commenced	Mode of inhalation not stated. Six drams of chloroform used. Not fully under its influence	Great excitement, with tossing about of limbs, followed by sudden collapse.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
62	June 26, 1858. 'Lancet,' vol. i, p. 630. Hôpital de Gros Caillou, Paris. Grenadier, æt. 45. Under the care of M. Ceccaldi	Castration for tuberculous disease of testis. Operation not commenced	Charpie in a fold of linen. Quantity not stated. Incomplete anæsthesia. Two to three minutes	Fresh chloroform was added, when patient suddenly sprang up, struggling to recover breath, and fell back, with strong expiration, motionless.
63	July 10, 1858. 'Med. Times,' p. 41. Reported in 'Macleod's Med. Surg. History of the Crimean War,' vol. ii, pp. 268-9. Case of Martin Hennessey, æt. 32. Crimea; 62nd Regt. (Occurred in August, 1855)	Removal of finger. Not commenced	A fold of lint. About two drams of chloroform used, given in 30-minim doses. Anæsthesia nearly complete	The chloroform caused a little cough at first, which soon ceased, and the stage of excitement set in. When nearly insensible he did not breathe freely, and there was spasmodic action of the larynx, as if from repeated swallowing of saliva. Chloroform was removed, and respiration had entirely ceased. No pulse could be felt. Artificial respiration and other means produced no effect. The heart's action continued for some time after the breathing and pulse had failed. *P.M.*—Frothy mucus throughout air-passages. Lungs healthy. Heart-substance healthy and firm; a little fat on external surface of left ventricle; valves natural. The chloroform was examined by Professor Maclagan, of Edinburgh. It was decomposed, evolved chlorine copiously, and had acid reaction from development of hydrochloric acid. There was no odour resembling chloroform, and it was acrid and nauseous when inhaled. "This chloroform was totally unfit for use. . . . . No one could inhale the stuff you sent me without having cough and bronchial irritation, probably spasm of the glottis, caused by it."
64	Sept. 11, 1858. 'Brit. Med. Journ.,' p. 780. Towcester. Reported by Mr. Watkins. Case of William Rush, æt. 11	To examine an injury of the foot, of some weeks' standing	Cotton handkerchief. Amount not stated. Extreme insensibility. Ten minutes from the commencement of inhalation	The boy was much frightened, and breathed irregularly at first. Fresh chloroform was added, and after six or eight inspirations he became insensible. The handkerchief was given to the mother to hold, and the examination of the foot commenced. Stertor set in, and the chloroform was discontinued. Pulse became imperceptible, lips livid. Artificial respiration produced a few short inspiratory efforts. No return of pulse.

65	Sept. 18, 1858. 'Lancet,' vol. ii, pp. 261-314; and 'Brit. Med. Journal,' Sept. 4, p. 753. Epsom. Servant girl	Extraction of tooth. Completed	Handkerchief. Administered by a druggist	"Seized with alarming symptoms." No further particulars.
66	Oct. 9, 1858. 'Med. Times,' vol. ii, p. 374. Ophthalmic Hospital, Moorfields. Male, æt. 8	Operation for strabismus. Commenced	Piece of lint. About 1½ dr. used at three applications. Incomplete anæsthesia	He flinched as the operation was commenced. More chloroform poured on lint. Face instantly became pale, and pulse ceased. Up till this had been quite regular, 80 per minute.
67	Oct. 16, 1858. 'Lancet,' vol. ii, pp. 410-457. Private case of Mr. Lawrence, at Barnes. Female, æt. about 45	Operation not mentioned	Handkerchief. Amount not stated. Ten minutes	No particulars.
68	Feb. 5, 1859. 'Med. Times,' vol. i, p. 142. Case of M. Richet, Hôpital St. Louis, Paris. Mechanic, æt. 43	For reduction of subcoracoidean dislocation of shoulder. Reduction effected	Piece of linen rolled into cone. From 4–5 drams used. Anæsthesia. Chloroform had been removed, and remarks were being made by operator	Excitement followed the fresh application of chloroform, but soon passed off. Operation completed. Pulse and respiration regular; when pulse suddenly stopped; a few rapid, deep inspirations continued.
69	Feb. 26, 1859. 'Med. Times,' vol. i, p. 220; 'Lancet,' vol. i, p. 202, Feb. 19, 1859. Case of M. Marjolin, at Hôpital St. Eugénie, Paris. Female, æt. 7½	Forcible straightening of contracted hip-joint. Not commenced	Sponge. About a dram. Incomplete anæsthesia	Resisted when touched. On second attempt crying and struggling ceased; circulation had stopped; lungs continued to act for a few respirations.
70	June 4, 1859. 'Med. Times,' vol. i, p. 581. Mr. Critchett. Royal Oph. Hosp., Moorfields. Female, æt. 15	Operation for strabismus. Just commenced	Piece of folded linen. Quantity not stated. Complete insensibility	She gave a shriek, and became insensible. The operation was commenced. Face became livid; pulse could not be felt; slight inspiratory efforts at intervals for a long time, and movement of nostrils, for at least half an hour, after cessation of pulse.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
71	July 23, 1859. 'Med. Times,' vol. ii, p. 81. Mr. Holt's case. Westminster Hospital. Male, æt. 45	To make incisions for extravasation of urine. Completed	Inhaler. One dram and a half used. Anæsthesia	The addition of half a dram of chloroform produced insensibility, and inhaler removed. Face became pallid. Pulse fluttered, and ceased. Respiration continued for one to two minutes.
72	Aug. 20, 1859. 'Med. Times,' vol. ii, p. 194. Mr. Solly's case. St. Thomas's Hosp. Male, æt. 28 (R. W.), intemperate	Amputation of foot for an old injury. Not commenced	Inhaler. One dram used. Not under influence. (Had been fully so for an hour and a half for a previous operation)	When about half a dram had been taken pulse suddenly failed; face pallid. Inhaler removed. Gave a few gasps, passed urine, and died.
73	Oct. 22, 1859. 'Lancet,' vol. ii, p. 412. "Dreadnought" Hospital Ship. Reported by Mr. Bedford. Male, æt. 24	To apply nitric acid to syphilitic sores. Application commenced	Cone of lint covered with oiled silk. About two and a half drams given at intervals. Imperfect anæsthesia. Dead in twenty minutes	Resisted the application of the acid. Struggling suddenly ceased; face pallid; pulse and breathing stopped. Artificial respiration produced a few inspiratory efforts.
74	Nov. 19, 1859. 'Med. Times,' vol. ii, p. 503. London Hospital. J. P. (male), æt. 57 (very intemperate)	For delirium tremens, following a fracture of tibia and fibula involving the knee-joint	Piece of lint. About half a dram. Not under influence	After two or three inspirations the man "writhed," and fell back dead.
75	Dec. 3, 1859. 'Lancet,' vol. ii, p. 576. Hôpital de la Charité, Paris; under care of M. Manec. Female, æt. 50	To reduce a dislocation of the shoulder. Completed	"A simple compress." Chloroform given in small quantities, and at intervals. Complete insensibility	Reduction effected. Chloroform removed. Face became congested. Respiration failed, then stopped. Death-like pallor. No pulse. Artificial respiration and galvanism caused inspiratory efforts, but no return of pulse.
76	Jan. 7, 1860. 'Lancet,' vol. i, p. 20. Alloa, Scotland. Case of D. Renwick, æt. 27	Evulsion of ingrowing toe-nail. Operation completed	A towel, held by himself. Perfect anæsthesia. (Had frequently taken chloroform before)	Did not recover from chloroform. Pulse and respiration at first natural; gradually became less frequent, till they ceased, in spite of artificial respiration.

77	June 9, 1860. 'Brit. Med. Journ.,' p. 443. Doncaster. Female.	To relieve neuralgic pains	Administered by her daughter, aged 10, upon a cloth. In the habit of taking chloroform in this way, very frequently, and in enormous doses	The last dose given at nine o'clock; found dead at ten. Supposed to be asphyxiated. Had taken it twice to insensibility on the same day.
78	July 28, 1860. 'Med. Times,' vol. ii, p. 83. Liverpool Workhouse. Female, old. (Lunatic)	To allay excitement	Quantity not stated. Given on lint by the master of workhouse. Dead in eight to ten minutes. Had frequently taken chloroform before	Great excitement and struggling.
79	Sept. 15, 1860. 'Brit. Med. Journ.,' p. 731. Bellevue Hospital, New York. Case of Michael Lanahan, æt. 40	Circumcision for chancres beneath prepuce. Not commenced	A napkin. An ounce and a half, used in small quantities. About five minutes	After inhalation for about four minutes a sudden stertorous expiration caused discontinuance of chloroform, and after a few laboured respirations the breathing ceased.
80	Sept. 29, 1860. 'Lancet,' vol. ii, p. 309. Northampton Infirmary, under the care of Mr. Mash. Case of P. Carroll, male, æt. 42 (intemperate)	To remove a fatty tumour from the back. Operation not commenced	Piece of lint. Five and a half drams. Complete insensibility. Eight to ten minutes	Chloroform suspended, and was rolled on side to facilitate operation, when stertor set in, with great congestion of face. Respiration and pulse ceased. Artificial respiration produced some inspiratory efforts.
81	Dec. 22, 1860. 'Med. Times,' vol. ii, p. 618. Private case of M. Faus, in Paris. Male, æt. 26	Removal of toe-nail. Operation performed	Cone of paper containing charpie. Quantity not stated. Anæsthesia	At first much excitement, and fresh chloroform twice added. The operation ended, and chloroform removed. A moan was heard. Face pallid. Pulse ceased. Artificial respiration caused faint inspiratory attempts; no pulsation.
82	Summer, 1860. Notes from Mr. Ralfe. King's College Hospital, under the care of Mr. Henry Lee. Male, about 55 (very intemperate)	Perineal section for old stricture of urethra. Operation just commenced	Inhaler. About one dram. Complete anæsthesia	Took chloroform quietly; breathing natural. Face turned pale, and pulse ceased. A few faint inspiratory efforts afterwards. None followed artificial respiration.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
83	June 29, 1861. 'Med. Times,' vol. i, p. 683. Reported by Dr. Dobbie. J. C., male, æt. 31 (drunkard)	To induce sleep in an attack of delirium tremens	Handkerchief. Two to three drams. Not fully under influence	Much struggling and excitement at first. Half to one dram added. After several inspirations suddenly fell back; the breathing much embarrassed. The handkerchief at once removed, and artificial respiration practised. Three inspirations followed.
84	July 6, 1861. 'Med. Times,' vol. ii, p. 22, from 'L'Union Médicale,' No. 75. Mauritius. Male, æt. about 35	Extraction of tooth. Completed	On handkerchief, by himself. Quantity not stated. Not completely under influence. Death in five minutes after extraction	A sudden movement of the head at the moment of extraction, followed by great turgescence of face and convulsions. These increased in severity until death.
85	Sept. 28, 1861. 'Med. Times,' vol. ii, p. 321. Cumberland Infirmary. Reported by Mr. Devereux. T. C., male, æt. 35	Introduction of catheter for retention of urine. Commenced	Lint in shape of cone. Two drams. Anæsthesia complete	Insensibility being produced, chloroform removed. In two minutes face changed; lips livid; pupils dilated; one deep inspiration, and pulse ceased. Artificial respiration caused two or three spasmodic inspiratory efforts.
86	Sept. 28, 1861. 'Med. Times,' vol. ii, p. 321. Mr. Annandale's case. Newcastle Infirmary. Male, æt. 32	Amputation of thigh for scrofulous disease of knee-joint. Operation not commenced, the tourniquet being applied	"A simple fold of bandage." Two drams. Under influence of chloroform	At first greatly alarmed, but readily yielded to influence of chloroform, without excitement. A sudden relaxation of the sphincters took place. Pupils dilated. Pulse ceased. The respiration continued for a few seconds.
87	Nov. 9, 1861. 'Med. Times,' vol. ii, p. 490. Brighton. Reported by Mr. Nourse. Male, æt. 50 (apparently intemperate)	Removal of piles by écraseur. Not commenced	A hollow sponge. Two to three drams. Complete insensibility	Much excitement and struggling at first. Forty to fifty drops of chloroform added, and, soon after, stertor caused withdrawal of sponge. The stertorous breathing increased. Face livid, dusky. Pulse ceased. Respiration gradually failed. Artificial respiration caused a few faint inspiratory efforts.
88	Nov. 16, 1861. 'Med. Times,' vol. ii, p. 519. St. Mary's Hospital. Male, æt. 8	Plastic operation on face to relieve deformity from a burn. Operation half completed	Inhaler. Fully under influence of chloroform	The inhaler had been removed for fully two minutes, to allow the operation to proceed. Sudden pallor of face occurred, and the pulse stopped. Breathing continued for at least ten minutes—at first spontaneous; afterwards faint efforts from artificial respiration.

89	Dec., 1861. 'Brit. Med. Journ.,' vol. ii, p. 649. University College Hosp. Male (intoxicated). Mr. Erichsen's case	"To reduce a fractured ankle"	No particulars	No particulars.
90	—— 1861. 'Gazette des Hôpitaux,' p. 8; from Perrin and Lallemand's 'Traité d'Anesthésie chirurgicale,' p. 338, obs. lxxv. Near Bordeaux. Male, æt. 40	Amputation of leg for recent injury. Not commenced	No particulars	The patient was trembling, and extremely frightened. The chloroform was held a long distance from his face. After four inspirations the pulse and breathing suddenly ceased.
91	—— 1862. London Hosp. Notes of case by Mr. Hutchinson. Male, under middle age	To reduce a dislocated humerus. Completed	No particulars. Under influence of chloroform	Took chloroform readily. Just as reduction was effected pulse stopped, and, after a few gasps, breathing also. Artificial respiration and galvanism produced no effect.
92	Jan. 12, 1862. 'Brit. Med. Journ.,' vol. i, p. 40. St. Mary's Hosp., under care of Mr. James Lane. Male, æt. 36	Forcible extension of contracted hip-joint. Not commenced	Inhaler. About a dram and a half. Not fully under influence	The man was highly nervous. One dram of chloroform being insufficient, half a dram more was added; and after a few inhalations the muscles became rigid, and he tried to raise himself in bed, when he suddenly fell back, the face became pale, and the pulse, which had been good up to this, stopped, the breathing became laboured, and shortly ceased. Artificial respiration of no avail.
93	Jan. 25, 1862. 'Lancet,' vol. i, p. 114. San José Hospital, Lisbon. Male, æt. 29. Occurred in 1859	Removal of cysts from eyelid. Not commenced	Piece of lint. About two drams. Not under influence	The inhalation caused great excitement, followed by collapse. The patient sneezed violently three times, and heart's action ceased. The breathing continued at intervals.
94	Feb. 22, 1862. 'Lancet,' vol. i, p. 204. General Hospital, Hobart Town. Reported by Mr. Turnley. Sailor, æt. 35	Castration for malignant disease. Operation not begun	"The usual way." Quantity not stated. Anæsthesia perfect. Four minutes	When insensibility set in the pulse became very feeble, and the chloroform was removed. In twenty seconds the heart stopped. Respired eight or ten times naturally. Artificial respiration ineffectual.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
95	May 17, 1862. 'Lancet,' vol. i, p. 534. London. Private case of Dr. Dieudonné. Male, æt. 33	Operation for fistula-in-ano. Not commenced	A piece of folded lint in a handkerchief. About one third of an ounce used. About ten minutes from commencement of inhalation	Was still quite sensible at the end of five minutes' inhalation. Soon after, he suddenly raised himself in bed, and the breathing ceased. No effect from artificial respiration.
96	June 28, 1862. 'Med. Times,' vol. i, p. 676. United Hospital, Bath. Female, æt. 40	Removal of tumour (cancerous) from lower jaw. Commenced	A napkin. About three drams in successive doses. Complete insensibility. Six or seven minutes	Anæsthesia complete in six minutes. The operation was commenced, and the third dram applied. Patient took one inspiration, and pulse stopped. The chloroform removed, and three or four gasps for breath followed.
97	Aug. 8, 1862. 'Med. Times,' vol. ii, p. 186. King's College Hospital, under care of Mr. Partridge. Female, æt. 17. Additional notes by Mr. W. F. Clarke	Plastic operation to close a large wound of labium, resulting from a sloughing ulcer. Completed	Inhaler. One dram and a half. Anæsthesia complete. About twenty minutes from commencing the inhalation	Was restless, and struggled much at first. After three or four minutes she suddenly became quiet and the breathing stertorous; lips and face pale; pulse stopped; heart's action imperceptible. She gasped at intervals for about fifteen minutes. Artificial respiration was employed.
98	Oct. 23, 1862. 'Med. Times,' vol. ii, p. 482. Painswick, Stroud, Gloucestershire. Mr. Cubitt's case. Male, æt. 23.	Amputation of thigh for old disease of knee-joint. Operation not begun	Inhaler. Quantity not stated. Insensibility	Some excitement produced at first, followed by a tranquil sleep. Was lifted from the bed to operating-table, when a sound as of vomiting was heard. Respiration and pulse ceased. Heart's action imperceptible. Laryngotomy and artificial respiration useless.
99	Oct., 1862. London Hospital, under the care of Mr. Adams. Male, æt. 23. Notes of case by Mr. Hutchinson. Patient very wasted and debili-	Amputation of the leg at the lower third, for disease of the bones of the foot. The limb was removed, and the arteries were being tied	Piece of lint. "A considerable quantity." Anæsthesia produced without anything unusual occurring	The man moved the limb slightly, and more chloroform was applied, when he suddenly became deathly pale, and his pulse ceased; "aspect like that of a corpse." The tongue drawn forward, and artificial respiration by compressing the chest kept up for half an hour. Efforts at inspiration occurred at intervals of from thirty seconds to a minute, for

	tated from disease, supposed to be phthisical			eight or ten minutes after the pulse had ceased—some of them of considerable force—so as to lead to a hope of a favorable issue; but no return of pulse or of colour to the face. Once or twice, with inspiration, a movement of the neck and right arm. Galvanism produced no effect beyond a quivering of the muscles. *P.M.* twenty hours after death. —Blood perfectly fluid, "like thin tar and water." Heart flabby, moderately full, no coagulum, valves healthy, muscular substance natural both to the eye and under the microscope. Lungs contained more air than usual, everywhere crepitant; did not collapse much on opening chest. No tubercle anywhere. Liver and kidneys normal.
100	Nov., 1862. 'Med. Times,' vol. ii, p. 548. Bellevue Hospital, New York. Female, young	To examine an injury of the shoulder. Under examination	Mode of administration and quantity not stated. Incomplete anæsthesia	No particulars.
101	Dec., 1862. 'Med. Times,' vol. ii, p. 669. Guy's Hospital, under the care of Mr. Bryant. Female, æt. 38	Plastic operation for vesico-vaginal fistula. Not commenced	Inhaler. Two and a half to three drams. Not fully under influence	After six or seven minutes spasm of the respiratory and other muscles set in; there was complete opisthotonos, and respiration was suspended. Face became pallid; lips livid; pulse imperceptible, although the heart's action continued feebly for some minutes. Artificial respiration and other measures failed to excite any inspiratory effort.
102	April 8, 1863. London. Notes of case by Mr. J. H. P. Staples. Female, æt. 29, highly nervous and hysterical	Extraction of tooth. Not commenced	Handkerchief folded in a cone. About one dram used. Partially under influence. Less than four minutes	Forty drops sprinkled on the handkerchief produced excitement after one and a half to two minutes' inhalation. Twenty more were added, and the excitement passed away. Pulse good; breathing calm; eye sensitive to touch. Suddenly the head fell to one side, face became pallid, pupils dilated, pulse ceased; she gasped a few times convulsively. Artificial respiration and galvanism produced a few inspiratory efforts, but no return of pulse.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
103	Sept. 23, 1863. London Hospital. Notes by Mr. Herbert Spencer. Male, æt. 42	Excision of the elbow-joint (following an injury a month previous). Operation not commenced	A piece of lint. Two drams of chloroform—the second dram applied four minutes after the first. Slightly under its influence	At first breathing regular and pulse somewhat quickened: about one minute after the second dram was applied the man struggled and tried to raise himself; the pulse suddenly failed, breathing continued for some minutes, and the lips remained florid. The tongue was immediately drawn forwards, artificial respiration and galvanism commenced, and continued for three quarters of an hour, but of no avail. *P.M.* examination.—Heart and right lung healthy; the left so infiltrated with tubercle as to render it almost useless for respiration.
104	Sept. 24, 1863. St. George's Hospital. Notes by Mr. Thomas Jones. Male, æt. 30; apparently a very healthy man	Operation for removal of necrosed portion of tibia. Commenced	Inhaler. About two drams. Complete anæsthesia. Death in six to seven minutes from commencing to inhale	Took the chloroform well; breathing regular and even, as also was the pulse. Operation commenced in five minutes; and a minute later the pulse, which was continuously watched, ceased instantaneously; the breathing continued unchanged for several seconds, when the face became pale, and the respirations lower, and soon stopped. Ammonia to nostrils, cold affusions, artificial respirations by compression of chest and by Silvester's method, galvanism to cardiac region and to needles passed into the heart, produced no result. *P.M.* twenty-three hours after death.—Brain dark, vessels congested, especially those of the cerebellum. Pericardium contained a quantity of turbid yellow serum. Heart; mark of one needle about an inch above the apex, the other an inch above this, both in left ventricle, which was partially contracted; right not so; valves healthy; slight fatty deposit amongst fibres of right ventricle, none on left side; to the eye the muscular tissue of the heart was perfectly healthy. Lungs healthy; slight congestion below and behind on both sides. Abdominal viscera normal, but rather congested. The blood was universally fluid.

105	Nov. 7, 1863. 'Lancet,' vol. ii, p. 547. Salisbury, under the care of Dr. Blackmore. Female (young)	Operation for fistula-in-ano. Completed	A handkerchief. About four and a half drams used, administered by himself, no assistant present. Complete insensibility	The patient inhaled the chloroform readily, and was fully under its influence in six to seven minutes. Dr. Blackmore then removed the handkerchief, and commenced the operation, which lasted two or three minutes. She had not then revived; the breathing became gradually slower, and ceased in about three minutes. Cold affusion, ammonia, galvanism, and Marshall Hall's "ready method" for artificial respiration, proved useless. *P.M.* examination.—Chest contained much fat. Heart of a light yellow colour, fatty matter mixed with the healthy tissue, very flabby, walls of left ventricle abnormally thick, of right very thin. Lungs very small, engorged with blood, showing on section "the black patches which indicate death from chloroform." Liver very large, encroaching on the thoracic cavity, so as to interfere with the action of both heart and lungs.
106	Nov. 7, 1863. 'Lancet,' vol. ii, p. 547. Private case of Mr. Gay. Female, æt. 16	Operation for hare lip. Not commenced	An inhaler at first, afterwards a piece of lint	Was seated in a chair. In three or four minutes "the spasms which precede the loss of sensibility set in," when the pupils suddenly dilated, face became pallid and lips blue; no pulse could be felt.
107	May 16, 1863. King's College Hospital. Notes by Mr. W. Fairlie Clark. Male, æt. 28; not good health, intemperate. On Jan. 10, 1863, he had been operated on, under chloroform, for an exostosis of the femur, without any ill effects from its use	Operation for fistula-in-ano. Not commenced	Inhaler. One dram and a half. Partially under influence. Eight to ten minutes	He breathed quietly for six or eight minutes, and then struggled violently. Had a sort of tetanic spasm; back curved, so as to rest on his head and heels. The chloroform was suspended during this excitement. Pulse regular, good. The inhaler was about to be reapplied, when the pulse in the temporal artery intermitted twice, and ceased. The body became ashy pale; patient gave three inspirations, each shorter than preceding, and breathing stopped; pupils natural. Cold affusion, ammonia to nostrils, artificial respiration by Silvester's method, and galvanism, were of no service. No heart-sounds could be heard. *P.M.* forty-eight hours after death.—Heart apparently healthy so far as muscular tissue was concerned; ruddy in colour; cavities dilated, and their walls thin. Lungs universally adherent; they contained some scattered tubercle and one small vomica. Liver large, pale, fatty. Kidneys natural.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
108	Sept. 21. Male, æt. 22. Private case of Mr. H. C. Johnson, at the New Hummums, Covent Garden. He expressed no dread of chloroform to Mr. Johnson, but always had a great horror of it, and possessed an idea it would kill him	Operation for phimosis. Prepuce removed	Weiss's apparatus. Quantity not stated. Partially under influence	Was a little nervous; pulse good. In about four minutes face became slightly congested, and struggled to rise in bed. Chloroform suspended, and operation commenced. Face became dusky and lips blue. Cold affusion to face and compression of ribs caused a deep inspiration, but pulse could not be felt. Artificial respiration, and other measures, were tried; he breathed at intervals several times, but respirations became fainter, and at length ceased. No *P.M.* allowed.
109	Probably in 1855 or 1856. Letter from Dr. Recordon, of Lausanne, to Dr. Marcet; received Nov. 20, 1863. Male, æt. 14–15, the son of Dr. de la Harpe.	Extraction of a tooth	No particulars	The patient was seated; he had syncope, and died.

TABLE B.—*Imperfect Reports of Fatal Cases.*

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
1	Sept. 18, 1858. 'Lancet,' vol. ii, p. 314. Dorking. Age and sex not given	Not stated	Apparently on a handkerchief	No particulars.
2	—— 1858. 'Medical Times,' p. 534	Another death referred to by Dr. Robert Lee from chloroform administered during labour, said to have occurred at Edinburgh, in the practice of Dr. Matthew Duncan	...	No particulars.
3	April 9, 1859. 'Medical Times,' vol. i, p. 376. Paris, Hôpital de la Pitié. M. Maisonneuve's case. Male, æt. 60	Reduction of dislocated hip. Completed	Mode of administration and quantity not given. "Never fairly under influence." Death in twenty minutes after the chloroform was removed	Very violent efforts necessary for reduction, which was at length accomplished, when symptoms of "cerebral congestion" set in.
4	Jan. 7, 1860. 'Lancet,' vol. i, p. 23. Liverpool. Male, age not stated	Suicidal	Purchased eight and a half ounces of chloroform. Appears to have poured it into a dish, and bent his head over it.	
5	April 21, 1860. 'Lancet,' vol. i, p. 412. Twickenham. Male, age not given	Supposed to be suicidal	A bottle containing a dram of chloroform was found on the table within reach of the body	The face and body were livid, cold, rigid. Bladder and rectum had voided their contents.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
6	May 4, 1861. 'Brit. Med. Journ.,' p. 477. Cincinnati, U.S. Male, age not given. Related in letter by Dr. Kidd	Operation for glaucoma. Apparently commenced	Not stated. Death in half an hour	Half an hour was occupied in administering chloroform, when vomiting set in, followed by a sudden fit and gasping for breath. The respiration ceased, but the pulse and heart "beat vigorously" for half an hour.
7	May 25, 1861. 'Brit. Med. Journ.,' vol. i, p. 549. Thornbury, Gloucestershire. Reported by Dr. Davey. Male, æt. 7	Tracheotomy for laryngitis. Completed	Not stated	Inhalation relieved the "spasm" (*sic*) of the glottis. The tube was readily inserted. Patient gave a gasp, and died. Artificial respiration ineffectual.
8	Dec. 14, 1861. 'Medical Times,' vol. ii, p. 625. Notting Hill Dispensary. Male, æt. 19	Probably suicidal	Was found with a cap containing a handkerchief placed over his face. The quantity used is not known, but about four drams had been taken from a bottle. Had frequently inhaled it before.	
9	—— 1860. 'New Sydenham Society Year-Book of Medicine and Surgery' for 1860, p. 463; from 'Virchow,' vol. xvii, pp. 5, 6. Related by Dr. Bückner. Male, æt. 40	To relieve the pain in passing gall-stones	Had suffered from the passage of gall-stones six to seven years, and to relieve the pain had taken morphia to the extent of twelve grains in the day, but had reduced it to about one grain and a half, and inhaled ether or chloroform during the paroxysms. Of ether he would use four to five pints, of chloroform from eight to thirty ounces, in a few days. He experienced relief, but subsequently resorted to laudanum. He had several attacks of mania, but was quite lucid in the intervals. "Dr. Bückner found him one morning in bed, breathing tranquilly, in the condition produced by long inhalation of chloroform." *P.M.* examination showed the organs unaltered. Gall-stones in gall-bladder; one the size of a bullet in bile-duct.	

TABLE C.—*Cases of Death which occurred some time after the use of Chloroform.*

No.	Date. Authority. Place. Name. Sex. Age.	History. Symptoms. Treatment.	Post-mortem appearances.
1	April 27, 1852. Reported by Dr. Parker, of Sunderland. Male, æt. 16	Had suffered from symptoms of stone for twelve months; previously very healthy. There was great difficulty in inducing anæsthesia, owing to the boy being frightened at the "doctor's coming to take the stone away." Nearly three quarters of an hour elapsed before he was under the influence of chloroform. The lateral operation was performed, there was difficulty in removing the stone, which was small, of uric acid. He was never perfectly conscious after the operation; during the night constantly delirious; felt pain when abdomen was pressed fourteen hours after operation, and died at the end of twenty-eight hours.	No post-mortem examination.
2	Dec., 1862. Reported by Mr. Tatum. Female, æt. (about) 28. A slight, fair, nervous woman	This patient took chloroform for the removal of a small mammary tumour. The mode of administration is not stated. "A very moderate quantity" is said to have been given. The operation was performed on December 27th, at 3 p.m. She was very sick and faint afterwards, requiring the use of stimulants. During the night she was very restless, notwithstanding the exhibition of morphia. At 5 a.m. on the 28th violent vomiting of thin biliary matter again set in, and continued for several hours; it was at length checked by a dose (gr. iij) of calomel, which, however, was soon followed by purging. This went on until night, and produced depression to an extreme degree; stimulants failed to rally her. She continued in this exhausted condition, although highly restless, until the afternoon of the 29th, when she became unconscious, and died at 11 p.m., fifty-six hours after the operation. For several hours before death her pulse was quite imperceptible. The wound had healed by first intention.	No post-mortem record.

No.	Date. Authority. Place. Name. Sex. Age.	History. Symptoms. Treatment.	Post-mortem appearances.
3	April 15, 1863. Reported by Mr. Francis Lloyd. Female, æt. 52.	Very stout and of plethoric habit, but extremely timid and nervous. Had been suffering from onychia of the left thumb for several weeks, but would not permit any incisions to be made. Under a fresh attack of inflammation she was persuaded to take chloroform for the purpose of operation. She was unusually long in yielding to the anæsthetic, and required a "more than ordinary proportion of chloroform vapour to atmospheric air;" she passed suddenly from a state of excitement to one of coma, which condition passed off in a minute or two, and nothing of an unusual character presented itself again, her pulse being better after conclusion of the operation than it was at the commencement. Rather less than one third of a grain of morphia was injected beneath the skin of the arm (she had taken about a grain in three doses during the night). The whole time from the first administration of chloroform until her return to consciousness was considerably under half an hour; she complained merely of feeling sick. She was left quite comfortable at half-past one. Until 3 p.m. she remained perfectly sensible, when she seemed to drop off to sleep, but soon after "began to breathe in a peculiar way." Her husband having examined her, and finding her pulse, as he thought, good, was satisfied, as he was accustomed to hear her breathe in this way when asleep. At 3.45 she was seen by her medical attendant, who was told that she was "sleeping nicely." He found her comatose, cheeks deep purple, lips livid, face cold, conjunctiva insensible, pupils contracted, not affected by light. Breathing stertorous, about three respirations in a minute; gurgling expiration; pulse 90, regular, small, and weak. Mustard poultices were applied to the epigastrium and calves of legs. Ammonia to nostrils and lips, followed by an enema of turpentine and coffee. Under this treatment the respirations increased to six or seven in a minute; the pulse, too, improved in volume and power, and the face grew less livid and dusky. Twelve leeches were also applied to the temples, but the slight improvement speedily gave way, the pulse became feebler, the respiration slower, till she sank gradually and died at 6 p.m., about five hours after the chloroform had been discontinued.	
4	—— 1860. New Syd. Soc. 'Year-Book of Medicine and Surgery' for 1860, p. 462. From 'Oest. Zeit.,' 1859, vol. v. Related by Dr. Linhart. Male, æt. 64	Chloroform was administered to a healthy peasant for the removal of a tumour of the eyelid; an ounce and a half was given in half an hour, and produced only slight drowsiness. He remained well till six o'clock the next morning, and suddenly died about eighteen and a half hours after the chloroform inhalation.	*Post-mortem examination*, twenty-four hours after death.—All the organs healthy. Coagulability of the blood slightly diminished.

TABLE D.—*Accidents with Chloroform, not fatal.*

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which accident took place.	Symptoms. Treatment.
1	April 17, 1858. 'Med. Times,' p. 416. New York. Male, young	Amputation of thigh. Ligature of last artery	No particulars. Chloroform very impure	Pulse and respiration suddenly stopped, countenance altered, and jaw dropped. Marshall Hall's "ready method" was persevered with for half an hour before there was any evidence of life; at end of forty-five minutes patient spoke.
2	June 28, 1858. 'Med. Times,' p. 662. New South Wales. Male	To examine the bladder and prostate gland	...	In three or four minutes the breathing was much accelerated, and the chloroform discontinued. The pulse ceased, and then the respiration. Heart sounds could not be heard. The "ready method" in two minutes caused signs of returning animation.
3	July 10, 1858. 'Med. Times,' p. 42. Paris, under care of Dr. Demarquay. Female, æt. 45	Removal of tumour from breast. Operation nearly completed	No particulars. Complete anæsthesia	More chloroform applied to prevent returning sensibility; pulse became weaker, and ceased; bleeding stopped, respiration suspended. Heart's action very imperfect. Face livid, then colourless; pupils dilated. Artificial respiration, *bouche à bouche*; in three minutes radial pulsation felt; in six or seven minutes complete recovery.
4	—— 1858. 'Lancet,' vol. ii, p. 106. Paris	Reference to a case in which artificial respiration proved successful in Paris for an overdose of chloroform.		

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which accident took place.	Symptoms. Treatment.
5	Nov. 6, 1858. 'Med. Times,' p. 481. Reported by Mr. Chas. Hunter. Female, æt. 18. Had frequently taken chloroform for the same affection, even two or three times a day	To relieve neuralgia of eyeball	Snow's inhaler	In one to one and a half minutes the patient moaned, and respiration ceased; pulse continued steady; shaking and cold affusion instantly employed. Pulse commenced to fail. Artificial respiration by compression, then the "ready method," which brought on inspiratory efforts, but it had to be persevered with for three hours. Mr. Hunter considers it a case of "secondary apnœa," due to a prolonged administration of chloroform ten hours before.
6	Jan. 1, 1859. 'Med. Times,' p. 9. Female, æt. 6. Had taken chloroform to insensibility three times before	Excision of knee-joint	Anæsthesia	Insensibility passing away, more chloroform given. Pulse became feeble, and in a minute ceased to beat; the breathing also stopped. The tongue drawn forward, and artificial respiration by compression tried, but for fifteen minutes no pulse or positive sign of life. An enema with brandy given. The pulse then became perceptible, but very faint and flickering; in ten minutes it improved a little, and then gasping took place. Ammonia applied to the nostrils caused a scream and sudden inspiration. Artificial respiration kept up for more than half an hour.
7	Mar. 12, 1859. 'Med. Times,' p. 271. Berlin, under the care of Professor Langenbeck. Male, æt. 58	Removal of tumour from back	Less than two drams used. Imperfectly under influence	Patient became restless. Face congested, breathing laboured; cold affusion and compression of thorax. Pulse regular, good; respiration became feeble, and ceased. The pulse stopped two minutes after the breathing. Insufflation through a catheter passed down the larynx of no service. Tracheotomy performed, and air blown into lungs through an elastic catheter. After six or eight inspirations by this means the pulse returned. Very weak and irregular, and again disappeared. As artificial respiration was persevered with, the pulse again returned and increased in strength and regularity. Cold water, friction, and galvanism also tried, but for an hour and a half life hung in the balance; then a paroxysm of coughing took place, expelling a quantity of bloody mucus. Unconsciousness, with dilatation of the pupils, continued, and it was not till after a long sleep that sensibility returned.

8	—— 1860. 'Medical Times,' vol. i, p. 196. France. Male, æt. 4	Removal of tumour from eyelid. Not commenced	A sponge. One dram used	In less than two minutes the pulse became feeble; inspiration short, rattling; face livid, limbs relaxed. Cold affusion, and ammonia to nostrils, mucus wiped from epiglottis with a sponge; friction of thorax. These tried for two or three minutes, when pulse ceased; pupils dilated; jaw dropped. Compressions of abdomen used for three minutes, when one conductor of Dubois-Raymond's induction apparatus was placed on the course of the phrenic nerve, the other on the seventh intercostal space, and alternated from side to side; this produced a sob, and on discontinuing the process there was a spontaneous inspiration; the pulse also returned. Compressions of abdomen was again resorted to, and continued for twenty minutes, when recovery was complete. Any cessation of the compressions caused enfeebled pulse and respiratory efforts.
9	—— 1860. 'Brit. Medical Journal,' p. 731. From 'American Medical Times.' America. Male, in a state of intoxication	Dislocation of femur. Reduction accomplished	A handkerchief	Attempts to reduce the dislocation without chloroform having failed, he was left for an hour to become sober. In five minutes after commencing to inhale, the respiration became slow, laboured, stertorous; the pulse flagged, skin livid, "patient apparently asphyxiated." The chest and buttocks were flapped with a towel, and cold affusion was applied from a height on the centre of the chest. In two or three minutes a deep inspiration took place. Chest then compressed. Perfect recovery in half an hour.
10	—— 1860. 'Lancet,' vol. ii, p. 429. Manchester Workhouse and Hospital. Female, æt. 23	Amputation of the thigh for old disease of knee-joint. Not commenced	Piece of lint. Two drams of chloroform. Anæsthesia	Inhaled chloroform quietly. In two or three minutes was insensible, with slow, steady pulse, and natural respiration. Suddenly the pulse and breathing ceased. Cold affusion and compressions of thorax applied; in two minutes there were feeble inspiratory efforts, which ceased when the compressions were discontinued. Tracheotomy was performed, and inflation of lungs by a catheter through the opening. Soon natural respiration commenced, and in an hour and a half she was removed to bed. In twenty days from this, chloroform was administered with an inhaler, when she took six drams. She died forty-seven days after the operation, of hæmorrhage. *P.M.* examination.—Surface of ventricles fatty, walls thin, valves natural; kidneys disorganized. The microscope showed advanced fatty degeneration of the heart and kidneys. This patient was extremely debilitated, and suckling a child nine months old.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which accident took place.	Symptoms. Treatment.
11	—— 1860. Reported by Dr. Hillier. Female, æt. 50. Private case of Mr. Quain	Removal of breast for cancerous disease. Not commenced	Inhaler. Insensibility not complete	In five minutes the conjunctiva was still sensitive, and pupil contracted; slight cough occasionally. A profuse perspiration broke out; the pulse failed, and, with the breathing, ceased. The pupils were "contracted to a pin's point;" face pale, lifeless. Cold affusion and compression of chest resorted to, when in about a minute the pulse could be felt fluttering, and soon began to improve. A gasp was given, and pulse rose at once, becoming regular, but weak. The operation was then performed, followed by faintness and vomiting.
12	—— 1861. 'Medical Times,' vol. ii, p. 540. Reading Hospital. Male, æt. 14	Operation for necrosis of tibia. Commenced	Anæsthesia	Was very timid, and struggled much at first. During the operation commenced again to struggle. Stertorous breathing set in, and the heart's action ceased; lips blanched; limbs flaccid. Artificial respiration, by Sylvester's method, was tried, with cold water to face and chest. In two or three minutes he gasped faintly. Galvanism was applied, and immediately excited the heart's action.
13	—— 1861. 'Brit. Med. Journ.,' vol. ii, p. 594. Salford Infirmary. Male, æt. 54	To pass a catheter in a case of stricture. Not commenced	Nearly under influence	The respiration became rapid, eyes assumed suddenly a corpse-like appearance. Pulse and breathing ceased. Tongue drawn forward. Artificial respiration by compression, cold water affusion induced several vigorous inspirations, and the pulse returned. The alarming symptoms lasted three minutes.
14	—— 1862. 'Brit. Med. Journ.,' vol. ii, p. 237. Norwich. Reported by Dr. E. Copeman. Male, young	To sound for calculus	Not fully under influence	Did not take chloroform readily. He breathed with stertor for a few seconds, and then respiration ceased. Tongue pulled forwards by means of a hook, and air was heard to rush into the now open glottis. Compressions of abdomen resorted to. Respiration and heart's action recovered.
15	—— 1862. 'Lancet,' vol. ii, p. 533. Guy's Hospital, under the care of Mr. Cock. Male, æt. 32	Removal of bulbous end of nerve from painful stump of arm. Operation commenced	Insensibility	Face suddenly became pale, pulse and breathing stopped. Cold affusion; tongue drawn forward; compressions of chest. In two or three minutes there was a sigh, and the pulse could be felt; in three or four minutes more, colour returned to cheeks, circulation and respiration were recovered.

16	Reported by Mr. Hine, of Nottingham. No date. Female, æt. 9	Removal of necrosed bone from femur. Commenced	Under influence	In thirty-five minutes face grew pale; pulse small, feeble, and fluttering; respiration slow and indistinct; surface of body cold, with relaxation of the sphincters. Exposure to cold current of air, warmth by heated flannels, administration of brandy and diluted liquor ammonia. She gradually, but slowly, recovered.
17	No date. Reported by Mr. Hine, of Nottingham. Male, æt. 64	Excision of a cancerous tumour from the axilla. Commenced	Complete anæsthesia	Had inhaled for ten minutes, when breathing grew stertorous, face congested, pupils rather dilated, pulse labouring and slow. These symptoms became more marked, and respiration seemed on the point of ceasing, when he was exposed to a current of air, and cold affusion employed. Gradually the danger passed away, but he continued quite insensible until operation was completed.
18	Nov. 22, 1863. St. George's Hospital. Reported by Mr. T. Jones, under the care of Mr. Holmes. Female (child). Had taken chloroform previously two months before	To examine a diseased elbow-joint	Inhaler. Two drams used. Anæsthesia in six minutes	After being insensible for about five minutes pulse became quick, then slow, and afterwards imperceptible; the breathing also ceased. Tongue drawn forwards; compressions of thorax resorted to; face became intensely livid. In two or three minutes spontaneous inspiratory efforts. Recovered in about ten minutes.
19	No date. Reported by Dr. Skinner, of Liverpool. Female, æt. 30	To relieve neuralgia of face	Napkin. Two or three drams. Had been under influence twenty minutes	Face changed to a pallid hue; eyes dull and glazed; jaw dropped. Respiration and pulse ceased. Heart sounds inaudible. Exposed to current of cold air. Tongue drawn forward, and in doing so a set of artificial teeth were found loose in the fauces, and removed. Artificial respiration by compressing thorax. In ten minutes there was a spontaneous effort at inspiration, and in ten minutes more had quite recovered.
20	No date. Reported by Dr. Parker, of Sunderland. Female, æt. 45	Removal of mamma for scirrhous disease. Completed	...	She was in the sitting posture. After having inhaled for one minute, the chloroform maintained a "strong influence" from 2 p.m. to 4 a.m. Fourteen hours after operation she suffered from syncope, the pulse being very irregular. She eventually recovered.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which accident took place.	Symptoms. Treatment.
21	—— 1852. Middlesex Hospital, under the care of Mr. Arnott. Reported by Mr. Sibley. Male	Strangulated hernia. Not commenced	Piece of lint in a handkerchief. About two drams, in three doses, were used. Under the influence	About forty minims were first given, with no effect beyond a slight spasm of the limbs, and a rise of pulse from 70 to 90. Thirty drops were added, but he still remained conscious at the end of three or four minutes, when forty minims were again added. In about half a minute the spasm relaxed. Pupils dilated; pulse 80, steady, full; slight stertor, and the chloroform was removed. The respiration became noiseless, slight, and in twenty seconds altogether ceased. The pulse intermitting. Cold water and a wet towel produced no effect, and the pulse ceased. Mouth to mouth insufflation, with pressure on the chest, produced a slight respiratory effort after about twenty inflations, and the pulse could be felt as a thread after seven or eight. Vomiting occurred, and respiration failed, but was restored by the same means, the pulse being weak, but steady. Soon vomiting again set in and the breathing gave way, but was reinduced by artificial respiration as above. Consciousness returned after a short time. He remained very drowsy, but was not allowed to sleep, as when he dozed the respiration diminished and the pulse faltered. He gradually recovered.
22	Oct., 1863. Middlesex Hospital, under the care of Mr. De Morgan. Communicated by Mr. Sibley. Female, æt. 39	Removal of breast for scirrhous disease. Completed	Snow's inhaler. About three drams used, in two-dram and one-dram doses. Perfect anæsthesia	After five minutes' inhalation she vomited some mucus, and the chloroform was temporarily discontinued, but at the end of fifteen minutes from its commencement she was completely under its influence, with muscles relaxed; pulse 80 (it had risen to 144); breathing easy, with slight stertor. Chloroform discontinued, and operation commenced. After four minutes sensibility was returning, and inhaler reapplied with another dram of chloroform; this soon produced its effect, and was again removed. The respiration now became more and more feeble, so as to be scarcely perceptible in three or four minutes, and pulse irregular. Operation was now completed. The face livid, and cold-water douche applied; the pulse and respiration improved slightly, but they shortly again failed, and there was an effort at vomiting. At this time (fifty minutes from commencing to inhale) the face was livid, hardly any respiration, and the pulse scarcely to be felt; the tongue was not retracted. Cold douche applied, and ammonia to the nostrils. In about a minute galvanism was employed; a few applications caused a deep inspiration, and in a few minutes breathing was fully restored. Galvanism was continued for a short time, and consciousness returned. She had no relapse, and recovered without a bad symptom.

23	—— 1860. New Syd. Society 'Year Book of Medicine and Surgery' for 1860, p. 463. From 'Virchow,' vol. xvi, pp. 5, 6. Related by Dr. Friedberg. Child, æt. 4	Removal of encysted tumour from eyelid	No particulars	Irritation of skin and compressions of abdomen produced no result. One conductor of Dubois-Reymond's induction apparatus applied over phrenic nerve at outer border of sterno-mastoid muscle, the other at seventh intercostal space on right and left sides alternately. This caused deep inspiration, with arching of the belly from contraction of the diaphragm; when interrupted, by way of experiment, there was a weak spontaneous inspiration. At the third application the countenance reddened, and the radial pulse became perceptible. When the induction stream was discontinued the pulse and respiration were much weaker, but were kept up by compression of abdomen, frictions, cold affusion, and ammonia at the nostrils. In twenty minutes the pulse and respiration were completely restored.
24	Jan. 16, 1864. 'Brit. Med. Journ.,' p. 63. Stamford. Related by Dr. Newman. Male	Suicidal	Did not know how much or how long he had inhaled chloroform. Did not swallow any	At 1·45 p.m. was found lying on a bed, partially undressed, with his face pressed in the pillow, congested, almost black; eyelids closed, pupils dilated, fixed; skin dark, moist with cold sweat; respirations six to eight per minute, irregular in force and rhythm; pulse 52, very feeble, regular. Breath smelt of chloroform. Cold affusion and friction of chest produced no effect. Artificial respiration by Sylvester's method did not increase the respirations. At 2 p.m. the stomach-pump removed a little glairy fluid. Water was injected and withdrawn; it smelt faintly, but distinctly, of chloroform (he declared afterwards that he had not swallowed any). At 2·5 p.m. galvanism—by Kemp's apparatus, of Edinburgh—was applied, one sponge over phrenic nerve at lower part of sterno-mastoid muscle, the other to the scrobiculus cordis, and occasionally rubbed over the chest. After several minutes the muscles were thrown into action. 2·15.—Respirations twelve per minute; pulse 65, stronger; heart's action not more evident; face less dark; no sensation. 2·40.—Face not so congested; respirations fifteen, deeper and more prolonged; pulse better, 65; pupils act from stimulus of light and remain more contracted, insensible to everything except he moved a little with the change of position of the sponge. 3·15.—Suddenly objected violently to the galvanism, and raised himself up in bed; recovered.

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler used. Amount of chloroform. Stage of exhibition at which accident took place.	Symptoms. Treatment.
25	May, 1848. 'Med.-Chir. Trans.,' vol. xxxiv, p. 43. St. George's Hospital. Related by Mr. Prescott Hewitt. Male, æt. 35	Excision of upper jaw for tumour. Six years' duration. Completed	Dr. Snow gave chloroform with inhaler, and afterwards on a sponge	Patient was seated in a chair, and when the operation was nearly completed became faint, but rallied, and the operation was completed. His pulse again failed. He was laid on the bed and "different restorative means" used. He recovered from the faintness a little, but his breathing became difficult. Laryngotomy was performed, and frothy blood escaped from the opening, but the patient soon died. At the *P.M.* examination the trachea and bronchial tubes, even to their minute ramifications, were found to contain a quantity of frothy blood. To this report is appended a letter from Mr. Snow, which contains the following paragraph:—"He seemed a good deal embarrassed with blood flowing into the throat, and leaned forward to get rid of it. I thought once he vomited some. When he became faint he was but little under the influence of chloroform, and no more was given. When taken from the operating-theatre there was no difficulty of breathing, and the influence of the chloroform had left him."

TABLE E.—*Cases where the Persons habitually inhaled immense doses of Chloroform.*

No.	Date. Authority. Place. Name. Sex. Age.	History.
1	Nov. 21, 1857. 'Med. Times,' p. 533. Reported by Dr. De Mérie. Male, æt. 30. Copied from 'L'Union Médicale,' Nos. 106 and 112.	This patient became so accustomed to the use of morphia that it failed to produced sleep, and he commenced to inhale chloroform to relieve the excessive restlessness from which he constantly suffered. He became addicted to its use, and continued it for three years, passing his nights and, at last, a portion of the day also under its influence. His countenance became altered, manners strange, and his "moral disposition impaired." For a time he was induced to restrain this habit, but he gradually returned to it, and indulged in the use of the drug for, it is supposed, about five years. He remained in bed till twelve or one o'clock, repeating the inhalations whenever he awoke. His appearance was that of a man given to drink. He used a pound of chloroform in five or six days; it caused no headache or uneasiness. On August 8th, having purchased some of the anæsthetic, and being probably under its influence, he fell from a railway carriage and broke both his legs. Double amputation was performed under chloroform, which readily produced insensibility. Morphia was given after the operation to relieve pain, but without effect, and at his urgent entreaties he was allowed to inhale chloroform, under the superintendence of a dresser, constantly, at short intervals. He died forty-two hours after the operation, completely exhausted. No autopsy was allowed. Decomposition of the body proceeded with "frightful rapidity."
2	—— 1857. 'Med. Times,' Nov. 21, p. 533. Reported by M. Vigla, in the 'Moniteur des Hôpitaux' for 1855, and copied from 'L'Union Médicale,' Nos. 106 and 112. Male, æt. 33	This man commenced to suffer from spasmodic asthma, for which he could obtain no relief until April, 1855, when his medical man advised him to try chloroform inhalations. This was successful, and he continued the inhalations of his own accord until he took enormous doses. Between May 1st and 10th he used fifty-two and a half drams. Jaundice now set in, and he diminished the amount during four days, but the paroxysms became so insupportable that on the 18th he used 112½ drams. At the beginning of June jaundice recurred, and he discontinued the anæsthetic, but the attacks of asthma increased and he renewed it, taking *daily* about forty drams of chloroform from the 4th to the 25th of June, when he entered a Maison de Santé. These large doses only slightly relieved him; he obtained but little sleep, although in a constant state of dulness, from which the paroxysms scarcely roused him. On admission his countenance was peculiar, eyes brilliant and moistened, expanded features, and smiling, having the appearance of a person "ravished with delight," or slightly intoxicated. Small quantities of chloroform were given him for two or three days, but delirium tremens set in and it was discontinued. The delirium lasted from June 27th to July 4th; his delusion for a part of the time was that he was in danger of poisoning from chloroform. On July 4th his breath still smelt of the drug, although he had not taken any since June 27th. His attacks of asthma ceased, and he left on August 8th.

Table F.—*Cases of Death from Swallowing Chloroform.*

No.	Date. Authority. Place. Name. Sex. Age.	History. Symptoms. Treatment.	Post-mortem appearance
1	Nov. 21, 1857. 'Med. Times,' p. 533. France. Related by M. Chereau. Male	Had long taken ether by inhalation to relieve neuralgic pains. Heavy losses affected his mind, and he purchased (it was afterwards ascertained) thirty-seven and a half drams of chloroform, with which he went to a hotel. Not being seen for twenty-four hours, the door of his room was forced, as a moaning was heard. He was lying on his back; face cadaverous; slow, regular respirations at intervals, with short gasps between; subsultus of muscles; pupils widely dilated, insensible; widely opened mouth, tongue dry like parchment; jugular veins distended; hands clenched; livid stains on body and limbs, resembling those on corpses, or as in persons asphyxiated with carbonic acid gas; heart still beating, but scarcely perceptible; pulse felt with difficulty; bladder distended, urine dribbling away. Current of cold air introduced; friction with hot cloths; mouth to mouth insufflation; strong coffee. After an hour patient could be roused to answer by repeated calling, but incoherently. At end of second hour still quite stupid and drunken, but looked about him. Pulse 50. Became restless; thirst excessive, violent pain in hypochondrium. Smell of chloroform with each expiration. He became conscious, and conversed rationally just before death, which soon occurred.	
2	April 30, 1859. 'Brit. Med. Journ.,' p. 354. London. Male, æt. 42	Left the room frequently at intervals, joined in the general conversation on his return, and appeared to be quite sober; suddenly uttered some incoherent remarks, sank back on the sofa upon which he was sitting, became insensible, and snored loudly. Pulse was scarcely perceptible; eyes dim and fixed, pupils contracted; face pale and livid; sweat-drops on forehead; breathing much embarrassed, entirely abdominal. Odour of chloroform with each expiration; this was at 7 p.m. Cold water dashed on chest and face. Ammonia occasionally to nostrils and galvanism were applied up to 1 a.m., when he was sleeping as if	Forty-four hours after death.—Very slight rigor mortis. Blood quite fluid. Cavities of heart engorged. Muscular substance pale and flabby, especially on left side. Lungs crepitant, congested at bases. Stomach contained half a pint of brownish fluid similar to

		under influence of alcohol, breathing heavily. At 9 a.m. an emetic was given of sulphate of zinc, followed by mustard and water. In about an hour and a half he vomited upwards of half a gallon of brownish blood and mucus, having a strong smell of chloroform, followed by copious purging of the same fluid. At 11 a.m. he was put to bed. Ammonia administered internally, friction with hot flannels, hot-water bottles to surface, and galvanism occasionally during the day, but the pulse gradually became imperceptible; respirations more feeble (abdominal), at long intervals, with puffing of lips; pupils widely dilated; surface livid and cold, until 8 p.m., when he died. At first there were occasional gleams of consciousness. At least five ounces of chloroform had been swallowed. He had been in the habit of taking alcohol, opium, and camphor, immoderately	that vomited, which smelt strongly of chloroform. Mucous membrane studded with red patches of congestion, the rugæ of fundus quite black. Lower part of œsophagus intensely reddened. Small intestine everywhere showed dark red spots; valvulæ conniventes looked like black bars across the bowel. Large intestine natural. Four pints of fluid (like that described above) in the bowel. Brain natural. Liver and kidneys large, pale yellow, from interstitial deposit.
3	—— 1859. 'Lancet,' vol. i, p. 400. London. Reported by Dr. W. P. Bain, Surgeon to the Poplar Hospital	On October 27th, 1858, found the patient, a female, in bed, lying on her right side; head dropped on the chest, countenance pale and death-like, mouth open; eyelids half closed, with the eyeballs rolled upwards, pupils slightly contracted, and just responded to the stimulus of light. Pulse 84, moderately full. The breathing abdominal, without stertor. Surface of body and extremities warm, but quite insensible. Some thin fluid of a yellowish colour had run from the mouth. In the room was a three-ounce phial, marked "Chloroform," with about half a dram remaining in it. The stomach-pump was used directly to inject about a pint of warm water, which was returned immediately, tinged with bile. Sinapisms were applied to the neck, chest, and feet. She continued insensible and motionless for several hours, except at intervals of about a quarter of an hour, when the angles of the mouth were drawn, with convulsive movements of the throat, and vomiting occurred with difficulty; the respiration also ceased for about thirty seconds, the lips and face becoming turgid and blue. This was succeeded by a long stertorous inspiration, and the breathing went on regularly again. The intervals of these attacks became gradually longer until about 1 p.m., when irritation of the conjunctiva caused her to move her hands and lips slightly. The pulse had risen to 100. At 3 p.m. she was more sensible. Pulse 100. The forearms and hands livid, rather cold; feet warm; she had	*P.M.* examination forty hours after death.—The skin of a yellowish-green colour, with much congestion of the depending parts. Abdomen tympanitic. Liver pale and finely mottled. Stomach with well-marked hour-glass contraction; surface much congested. On opening it the internal coat was highly inflamed, the rugæ larger than usual, and pulpy. For about two inches round the œsophageal opening the mucous and muscular coats were entirely eroded, the peritoneum alone remaining; around this erosion the tissues were extremely vascular. A similar change had occurred about the pylorus.

No.	Date. Authority. Place. Name. Sex. Age.	History. Symptoms. Treatment.	Post-mortem appearances.
3—	Case (*continued*).	vomited much bile. 6 p.m.—Pulse 120. She answered questions, complained of tenderness at the epigastrium. Had passed fæces and urine voluntarily, the latter in very large quantity. The odour of chloroform was very strong in the breath. Half-past 10 p.m.—Pulse 160. Skin warm, no headache, thirsty and sleepy. Stated that, in consequence of domestic troubles, she rose at 3 a.m., and poured a wineglassful of chloroform into a tumbler with an equal quantity of water, drank it, put out the candle, and went to bed. The narcotic effects of the drug had now entirely passed away. She suffered from symptoms of acute gastritis, and died on the eighth day. The odour of chloroform was present in the breath thirty-six hours after taking it.	Œsophagus natural. Kidneys the seat of old disease. Heart and lungs healthy.
4	—— 1862. 'Med. Times,' vol. i, p. 478. Translated by Dr. Wm. D. Moore, from the 'Hygiea' for February, 1862. Reported by Dr. Axel Lamm, Hon. Mem. Swed. Loc. Phys.. Case of male, æt. 35. It was afterwards ascertained that this gentleman was accustomed to inhale and swallow chloroform to relieve his restlessness, but was very sensitive to it by inhalation, half a dram having caused insensibility a few months previous for the purpose of having	At a quarter past 7 a.m. on October 7th it was found that the patient, being unable to rest, had taken a dose of chloroform about half-past 12 a.m., and was still sleeping, but so profoundly as to cause uneasiness. He was in an apparently tranquil slumber, with a rather hurried and audible respiration; the pulse full, slower than usual; skin warm, eyelids closed, pupils highly dilated and quite insensible. A smell of chloroform pervaded the room and was perceptible in the patient's breath. Face pallid. A bladder of ice applied to the head, cold affusions along the spine, and an enema given. At half-past nine more pallid, getting cold, breathing not so loud, pulse weaker and slow. Electricity was now used, one pole placed on the pit of the stomach, the other along the course of the phrenic nerve on either side. After a few applications, with an interval of several minutes between each, the respiration became somewhat deeper and more distinct. The electric current was also applied to the spine, thoracic muscles, and calves of the legs. An enema with twelve or fifteen drops of ammonia was given and retained. The tongue was drawn forward, and then hung cold, pale and dry, beyond the teeth. Iced water to chest and pit of stomach alternately with warmth. Occasionally air was blown in along a tube with the natural inspirations. Half-past 11 a.m. — The narcotism continued, but the pupils began to contract; the breathing improved, pulse fuller, heat of body increased. Frequency of respiration very great, particularly when compared with the pulse. Smell of chloroform distinct in expira-	*P.M.* examination thirty-eight hours after death.—Rigor mortis present. General congestion of the brain. Cerebral substance everywhere loose, soft, and dotted with blood. Dura mater adherent to skull. Small bony masses of falx major engorged with fluid blood, with a few coagula. Slight yellowish effusion between convolutions. Heart substance dark, otherwise natural. A small loose dark coagulum in right auricle. Lungs congested; the bronchi contained a quantity of frothy, bloody fluid, which extended into trachea and larynx. Stomach contained about three ounces of thick grayish fluid, in which floated a number of "gelatinous flakes." The cardiac

a tooth extracted. The quantity of the fatal dose was not accurately known. The bottle from which it had been poured might hold about two and a half ounces, and the wife thought it was nearly half full	tion. 5 p.m.—Abdomen distended with gas. No urine having passed, the catheter was introduced, but no water drawn off. Half-past 6 p.m.—Skin warmer than usual, moist; pulse full, rather quick. An enema with colocynth drops given, in about an hour it brought away a mass of grayish excrement. At 9 p.m. the patient began to breathe in a moaning manner. The eyes commenced to move, the pupils appeared sensitive to light; moved the head away when ammonia was applied to the nostrils; the eyelids also showed some sensibility, but there was none in any other part. Profuse perspiration slowly set in; pulse 160, and rather hard. The patient sat up occasionally, and looked around with an air of surprise, but speedily fell back again. His expression at times was quite sensible. This continued for about half an hour, when he got more and more restless, throwing his head backwards and forwards; skin soaked in perspiration; pulse weaker and more rapid. Mucus accumulated in the throat, and he died at 11·45 p.m., about twenty-three hours after drinking the chloroform.	extremity was much congested, and studded with numerous minute livid spots. The mucous membrane near the pylorus was corrugated, but pale. There were no erosions or circumscribed patches of inflammation. Kidneys, spleen, liver, normal Gall-bladder contained fluid green bile. No smell of chloroform when any of the cavities were opened.

TABLE G.—*Cases of Swallowing Chloroform, not fatal.*

No.	Date. Authority. Place. Name. Sex. Age.	Symptoms and Treatment.
1	Nov. 28, 1857. 'Med. Times,' p. 559. From 'Amer. Journ. of Med. Science' for Oct., 1857, p. 367. Dragoon	Drank two ounces of chloroform, and when seen, fifteen minutes after, had vomited, but was then quite insensible. The breathing was stertorous; pulse 60. The stomach-pump was used, and some aromatic spirits of ammonia injected. The pulse and respiration became slower and more feeble. The pupils insensible. Cold douche to the head did some transitory good, but the surface of the body became gradually colder, so the patient was wrapped in blankets, a stimulating enema given, and mustard poultices applied. He continued to get worse, the face became purple, the pulse intermittent and scarcely perceptible. In about two and a half hours after taking the chloroform slight improvement commenced. Four hours from this, sensibility had returned. He had great irritability of the stomach for several days, and eventually an attack of jaundice.
2	Dec. 12, 1857. 'Med. Times,' p. 615. Reported by Mr. H. D. Dean, of H.M.S. 'Indomitable.' Female, æt. 22	In a moment of excitement swallowed half an ounce of chloroform. When seen, five minutes afterwards, was generally convulsed, insensible, face flushed, pupils dilated, jaws clenched, foaming at mouth. Pulse full, oppressed. She was exposed directly to a current of fresh air, and the stomach-pump used, after which brandy and water was given every two or three minutes. She vomited, and became more sensible and less convulsed. The fluid ejected smelt strongly of chloroform. Ammonia was applied to the nostrils, and the cold douche to the head. In less than twenty minutes she was sensible, the convulsions had ceased, pulse regular and full. The brandy was continued at intervals. She had several relapses, becoming convulsed, with a clammy sweat and irregular flickering heart action, but ultimately recovered, with slight gastric and pharyngeal irritation.
3	—— 1861. 'Brit. Med. Journ.,' vol. i, p. 377. New York. Reported in 'Amer. Med. Times,' by Dr. Fennell. Female, æt. 18	She swallowed an ounce of chloroform, walked the length of the room, placed the phial on the mantelpiece and fell heavily to the floor. She was seen about twenty minutes afterwards, lying on a sofa, with a hot burning sensation at the stomach, countenance pallid, extremities cold, pulse feeble and quick. She refused to take any remedies, but an ounce of powdered ipecacuanha in warm water was forced down her throat. In a few minutes she commenced to vomit, the odour of chloroform being very distinct in the ejecta. About forty minutes after drinking the chloroform she became comatose, with stertorous breathing, feeble rapid pulse; contracted pupils, insensible to light and touch. Mustard poultices were applied to extremities, cold water to the face, and gentle flagellation with a towel. She remained insensible for about half an hour, when consciousness slowly returned. As this took place she passed through the various stages observed in persons who inhale chloroform. At 11 p.m., three hours after taking the drug, she had perfectly recovered from its effects.

4	—— 1862. 'Med. Times,' vol. i, p. 577. Reported by H. M. D. S. Male, æt. 50	On April 6th, at 8 a.m., he swallowed two ounces of chloroform. As he had been unwell for some days, he was not disturbed till 3 p.m., when he was found in a state of deep coma, the breath smelling strongly of chloroform. The pupils were widely dilated, quite insensible. Pulse slow and feeble. Surface of body colder than natural, movements of thorax scarcely perceptible. No sensation whatever. Sinapisms were applied, hot-water bottles, and cold affusions, but without any effect. The stomach-pump was now used, and removed a quantity of chloroform, mucus, and watery fluid. The viscus was thoroughly cleansed with warm water. Signs of consciousness soon returned, and in less than an hour he answered rationally. For three or four days a burning sensation was felt in the throat and epigastrium, which gradually passed off, leaving no ill symptoms of any kind.

TABLE H.—*Cases of Death from Chloric Ether.*

No.	Date. Authority. Place. Name. Sex. Age.	Nature and stage of operation.	Inhaler and amount of chloroform. Stage of exhibition at which death occurred, and time.	Mode of death.
1	Aug. 22, 1857. 'Med. Times,' p. 199. America. Male, æt. 5. Reported in 'Amer. Journ. of Med. Science' for July, p. 284	Removal of fatty tumour from the back. Tying the arteries after excision	A hollow sponge. One dram of mixture of four parts of washed ether and one part of chloroform. Complete anæsthesia	Vomiting set in. The pulse, which was previously good, suddenly ceased. He had lost four or five ounces of blood.
2	Aug. 14, 1858. 'Med. Times,' p. 174. United States. Dragoon, æt. 23 (very intemperate)	Examination of injured elbow. Not commenced	A sponge in a bell-glass, and afterwards a towel. Quantity not stated. The chloric ether, as used in the U.S. army, consists of one part of chloroform and two parts of absolute alcohol. The same bottle produced "unpleasant symptoms" in six other cases. Never completely under influence.	Excitement followed the application of the towel. Face became turgid, and the muscles relaxed. Vomiting set in. The pulse, which had been 100 per minute, suddenly stopped; but there were two or three inspirations after this. A piece of cabbage was removed after death from the larynx.

Appendix C.

SELECTED EXPERIMENTS FOR RESUSCITATION.

Artificial Respiration.—

From *Chloroform*—

After cessation of respiration.

Successful	(39) 10 per cent.	Recovery after 60 sec.
	(82) Chlor. strong.	Recovery after 75 sec.
	(88) Chlor. strong.	Recovery after 60 sec.
Unsuccessful	(67) Strong.	Death after 45 sec.
	(19) 5 per cent.	Death after 60 sec.
	(13) 10 per cent.	Death after 1 min. 30 sec.

After cessation of heart's action.

Successful	(66) Strong.	Recovery after 30 sec.
	(70) Strong.	Recovery immediate.
	(71) 4 per cent.	Recovery after 10 sec.
Unsuccessful	(68) Strong.	Death immediate.
	(69) Strong.	Death immediate.
	(72) 5 per cent.	Death after 15 sec.

From *Mixtures*—

After cessation of heart's action.

Unsuccessful	(65) C, strong.	Death after 10 sec.
	(73) C, strong.	Death after 15 sec.
	(74) A, strong.	Death after 10 sec.
	(75) A, strong.	Death immediate.

Galvanism.—

After cessation of heart's action.

Successful	(76) Mixture A, strong.	Recovery after 25 sec.
Unsuccessful	(77) Chloroform, strong.	Death after 10 sec.
	(78) Mixture A, strong.	Death after 10 sec.
	(79) Mixture A, strong.	Death after 15 sec.
	(80) Chloroform, strong.	Death after 10 sec. (Secondary asphyxia.)

Galvanism (continued).

	(81) Chloroform, strong.	Death immediate.
	(84) Chloroform, strong.	Death after 20 sec. (Secondary asphyxia.)
	(85) Chloroform, 7½ per cent.	Death after 5 sec.
	(87) Chloroform, 7½ per cent.	Death immediate.
After cessation of respiration.		
Successful.	(83) Chloroform, strong.	Death after 60 sec.
Unsuccessful.	(86) Chloroform, 7½ per cent.	Death after 60 sec. (Secondary asphyxia.)

Artificial Respiration with Oxygen Gas.—

(89) Chloroform, strong.	Recovery 60 sec. after cessation of respiration.
(90) Chloroform.	Recovery 90 sec. after cessation of respiration.

Artificial respiration, after cessation of respiration. Recovery.

(Exp. 89.) Chloroform, 10 per cent., was given to a very large dog. He became insensible at 3 min. 20 sec. At 6 min. 20 sec. the pulse suddenly stopped, and the respiration ceased. After 60 sec. artificial respiration was commenced by pressing the chest. At 8 min. 45 sec. there was an effort at respiration. At 11 min. 30 sec. the animal commenced to respire; the pulse in the femoral was perceptible. At 12 min. 30 sec. the cornea became again sensitive, and shortly after this he had completely recovered.

Artificial respiration. Recovery.

(82.) Chloroform vapour, of the strength of 8 per cent., from Mr. Clover's bag, was given to a rather large dog. He became insensible at 1 min. 5 sec. At 2 min. 20 sec. the respiration had nearly ceased; at 2 min. 45 sec. the last

effort at respiration took place. At 4 min. artificial respiration was commenced, the needle in the heart indicating a feeble but still regular movement of that organ. At 6 min. 45 sec. an effort at voluntary respiration took place, and after this natural respiration continued. At 7 min. 50 sec. the cornea became sensible, and the animal recovered.

Artificial respiration. Recovery.

(88.) Chloroform, $7\frac{1}{2}$ per cent., from Mr. Clover's bag, was administered to a small dog. At 1 min. he was insensible. At 6 min. 40 sec. the respiratory movement ceased. At 7 min. 40 sec. artificial respiration was commenced. At this moment the heart was still beating regularly, and the pulse could be detected in the femoral artery. At 9 min. 30 sec. there were some natural efforts at respiration, and soon afterwards the animal commenced to breathe freely.

Artificial respiration, after cessation of respiration. Unsuccessful.

(67.) Strong chloroform was given to a dog by means of a handkerchief. He became insensible at 1 min. 30 sec. The respiration ceased at 3 min. 45 sec. At 4 min. 30 sec. artificial respiration was commenced by means of the hands; the heart was at this time acting feebly. At 8 min. 30 sec. slight tremor was observed in the tongue and at the angles of the mouth. At 10 min. 35 sec. there were some efforts at respiration, and the artificial movements were interrupted. At 11 min. 30 sec., the natural respiratory movements having failed, artificial respiration was again commenced. These movements were not very effectual, owing to an accumulation of mucus in the fauces. There were no further efforts at respiration, or other signs of life. At 25 min. 30 sec. the artificial respiration was finally discontinued.

(19.) Chloroform, 5 per cent., was given by the trachea

to a small terrier dog. The heart's action became very uncertain and irregular. The respiration ceased at 1 min. 45 sec. Artificial respiration by pressing the chest was commenced at 2 min. 45 sec. There were slight efforts at natural respiration at 3 min. 30 sec.; but these ceasing, the artificial respiration was again commenced at 5 min. 5 sec. There was no further sign of life, and the artificial respiration was discontinued at 7 min.

(13.) Chloroform, 10 per cent., was given by the trachea to a moderate-sized bitch. The pulse ceased at 1 min. 15 sec. The respiration soon after ceased for a few seconds, but again commenced; it finally ceased at 4 min. 30 sec. At 4 min. 45 sec. the heart fluttered, but continued to beat. At 6 min. artificial respiration was commenced. At 7 min. there were some gasping efforts at respiration, and the artificial respiration was discontinued till 9 min., when, the natural efforts ceasing, it was again commenced. The movements of the heart ceased at 11 min. 45 sec., and after this there was no further sign of life. The artificial respiration was discontinued at 13 min. 30 sec.

Artificial respiration, after cessation of heart's action. Recoveries.

(66.) Strong chloroform vapour was given to a moderate-sized dog by a towel. He became insensible at 1 min. 15 sec. At 1 min. 30 sec. the pulse ceased to be felt in the femoral artery. At 1 min. 45 sec. the respiratory movements ceased; and at this time no movement of the heart could be detected by auscultation. At 2 min. 15 sec. artificial respiration was commenced. At 2 min. 50 sec. slight muscular movement was observed. At 5 min. 15 sec. some movement was noticed in the epiglottis. A few seconds later there were voluntary efforts at respiration, and the animal soon recovered.

(70.) Strong chloroform was given to a small dog by means of a towel. The animal became insensible at 1 min.

20 sec. At 2 min. 5 sec. voluntary respiration had nearly ceased. A pin was inserted through the walls of the chest, so that the movements of the heart could be observed. The last respiratory movement was at 2 min. 35 sec. The needle ceased to indicate any movement of the heart at 4 min. 50 sec. Artificial respiration was at once commenced. At 5 min. 35 sec. some movements of the tongue and of the tail were observed. At 6 min. 5 sec. there was movement of the epiglottis, and at 7 min. 20 sec. there were efforts at respiration. Shortly after this, breathing was freely established, and the animal recovered.

(71.) Air charged with 4 per cent. of chloroform vapour was given to a full-grown terrier dog. This was inhaled for 38 minutes without any symptoms of impending death. The animal was allowed partly to recover, and was then made to inhale air containing 5 per cent. of chloroform vapour. He became insensible in 1 min. The respiration ceased at 17 min. 15 sec.; and the needle ceased to indicate any positive movement of the heart at 18 min. At 18 min. 10 sec. artificial respiration by the hand was commenced. At 18 min. 40 sec. some gasps took place, but, being inefficient, the artificial respiration was continued till 24 min. 10 sec., when the animal breathed freely; it was then discontinued, and the animal recovered.

Artificial respiration, after cessation of heart's action. Unsuccessful.

(68.) Strong chloroform was given to a small dog by means of a towel. He became insensible at 2 min. 15 sec.; the respiration ceased at 3 min. 35 sec. The heart ceased to beat (as shown by the needle) at 4 min. 30 sec., but for some little time previously the movements had been very irregular. Artificial respiration was commenced immediately upon the heart ceasing to move (4 min. 30 sec.), and it was continued for 11 minutes (till 15 min. 30 sec.); the animal, however, exhibited no sign of restored anima-

tion. In this case there was some doubt whether the air passed into the chest during the artificial respiration.

(69.) Strong chloroform was given to a rather large dog by means of a towel. The respiration finally ceased at 4 min. 15 sec.; the movement of the heart, as indicated by the needle, at 5 min. 5 sec. Artificial respiration (manual), but without success.

(72.) Chloroform, 5 per cent., was given to a middle-sized dog. He became insensible at 4 min. 15 sec. The respiration ceased at 27 min. 20 sec.; the heart at 30 min. 15 sec. Artificial respiration commenced at 30 min. 30 sec., and continued for ten minutes, but the animal exhibited no further sign of life. In this case the respiration had been shallow and interrupted some little time before its cessation, and had been again renewed. The heart's action, also, for some time before its final cessation, had been very feeble and irregular.

Galvanism, after cessation of heart's action. Recovery.

(76.) A large dog was made to inhale the Mixture A by means of a towel. He became insensible at 2 min. 15 sec. The heart stopped at 10 min. 30 sec. (as ascertained by the needle); there was one respiration at 10 min. 35 sec. At 10 min. 55 sec. galvanism was applied—the one pole to the needle in the heart, the other to the mucous membrane of the prepuce, the current being slight in force and interrupted. At 11 min. voluntary efforts at respiration commenced. These movements of respiration continued regularly till 11 min. 50 sec., when they ceased; during this period no galvanism was applied. The needle did not indicate any movement of the heart. Galvanism was then again employed, and at 13 min. 50 sec. the animal again commenced to breathe, and some movement of the heart was observed. At 15 min. 30 sec. the respiration was regular, the heart beating 140 per minute. The galvanism was

gradually discontinued. At 18 min. the animal was breathing regularly. At 20 min. the cornea became sensible, and the animal recovered.

Galvanism, after cessation of heart's action. Unsuccessful.

(77.) Strong chloroform (by means of a towel) was given to a dog. He became insensible at 1 min. 30 sec. At 2 min. 30 sec. the respiration became very shallow, and the heart's action very irregular. The respiration ceased at 6 min., the heart's action at this time being slow and irregular (about 10 in 30 sec.). After this the heart's action became more regular for a short time. At 7 min. 15 sec. the needle indicated that the action of the heart had ceased for a few seconds; but it again went on beating. The heart seemed likely to stop again two or three times, and at 7 min. 10 sec. it ceased to move. At 7 min. 20 sec. galvanism was applied in the manner before described—one pole being connected with the needle in the heart, the other with the prepuce. The galvanism was continued for ten minutes, but the animal exhibited no further sign of life.

(78.) The Mixture A was administered to a dog by means of a towel. At 2 min. 15 sec. he became insensible. At 13 min. 50 sec. the respiration and heart's action ceased simultaneously. At 14 min. galvanism was applied in the manner before described, the current being sent from the needle in the heart to the prepuce. At 15 min. the heart again commenced to beat with some regularity. At 16 min. 45 sec. the heart was acting feebly, and only when the galvanism was applied. Soon afterwards all movement ceased; there was no effort at respiration, or any other sign of life. The galvanism was continued till 22 min. 30 sec.

(79.) The Mixture A was given to a rather large dog by means of a towel. At 4 min. 30 sec. he was insensible. At 16 min. 30 sec. the respiration became slow, feeble, and irregular (about 8 in 15 sec.); the heart's action at this

period being regular (25 in 15 sec.). At 19 min. 30 sec. the respiration ceased till 20 min. 45 sec., when a few respiratory movements again took place. In this interval the pulse in femoral artery ceased, and the heart's action became very feeble. At 21 min. 45 sec. the heart's action almost ceased, but slight movements were observed till 22 min. 30 sec. At 22 min. 45 sec. galvanism was applied as in the other cases—one pole being connected with the needle in the heart, the other with the prepuce, and subsequently with the mouth. There was, however, no further sign of life, and the galvanism was discontinued at 36 min.

(80.) Chloroform in a strong form was given, by means of a towel, to a moderate-sized dog. He was insensible at 2 min. At 3 min. the heart's action was irregular and feeble. At 5 min. 30 sec. the respiration ceased till 6 min. 30 sec.; when it was resumed it was extremely shallow at first, but subsequently improved. At 14 min. 30 sec. the respiratory movement finally ceased, and at 14 min. 55 sec. the heart stopped beating. At 15 min. 5 sec. galvanism was applied as in the other cases, from the needle in the heart to the prepuce. At 16 min. 15 sec. voluntary respiration commenced, and continued for rather more than a minute; gradually, however, it became more and more feeble. During this time there were no pulsations of the heart. At 17 min. 45 sec. there were a few pulsations of the heart, and at 18 min. 15 sec. these had quite ceased. At 25 min. 15 sec. the galvanism was discontinued, the animal exhibiting no further sign of life.

(81.) Chloroform was given in a strong form, by means of a towel, to a rather large bitch. At 1 min. 50 sec. she was insensible. At 3 min. 30 sec. the heart had nearly ceased beating. At 3 min. 50 sec. the respiration temporarily ceased, and the pulsations in the femoral artery were arrested. At 6 min. 45 sec. there were a few efforts at respiration, which ceased after a few seconds; the heart's

movement became extremely feeble, being scarcely perceptible; and all movement finally ceased at 8 min. Galvanism was at once applied as in the other experiments (from the needle to the prepuce), but the animal exhibited no further sign of life. At 19 min. 30 sec. the galvanism was discontinued.

(84.) Chloroform, of the strength of 8 per cent., was given, by means of Mr. Clover's bag, to a small terrier dog. He became insensible at 1 min. 15 min. At 4 min. 15 sec. the respiration was very shallow, the pulse very weak in the femoral. At 10 min. 35 sec. the respiration, the movement of the needle in the heart, and the pulse in the femoral artery, ceased simultaneously. At 10 min. 55 sec. galvanism was applied, the current was first directed from the needle in the heart to the prepuce, and afterwards from a needle in the diaphragm, the other pole being connected with the tongue. At 12 min. respiration commenced, and the pulse returned in the femoral; the galvanism was discontinued. Only a few efforts at respiration were, however, made, and these having failed, at 13 min. a few artificial respirations were made, and the galvanism was recommenced. The galvanism was continued till 17 min., but the animal did not exhibit any further sign of life.

(85.) Chloroform, of the strength $7\frac{1}{2}$ per cent., was given, from Mr. Clover's bag, to a middle-sized terrier bitch. The animal became insensible at 2 min.; at 9 min. 15 sec. the respiration ceased; at 9 min. 45 sec. the needle in the heart stopped moving. Galvanism was applied 5 min. after—one pole being placed on the neck, the other connected with a needle inserted in the diaphragm. A few seconds later slight movements were observed in the needle in the heart, but these soon ceased. There was no effort at respiration, or any further sign of life. The galvanism was discontinued at 13 min.

(87.) Chloroform ($7\frac{1}{2}$ per cent.) was given to a small dog, from Mr. Clover's bag. At 1 min. 30 sec. he was insen-

sible. At 9 min. 40 min. the heart ceased moving, its pulsations having been very feeble for some time. The respiration had nearly, if not quite, ceased, having been slight and irregular for some little time. Galvanism was at once applied—one pole being placed on the neck, the other attached to a needle in the diaphragm. At 10 min. 10 sec. the heart was again beating, and at 10 min. 30 sec. the respiration recommenced. At 11 min. 30 sec. the respiratory movement and the heart's action both stopped, and did not recommence. During the time that the heart's action was partially restored no pulsation could be detected in the femoral artery. The galvanism was discontinued at 16 min.

Galvanism, after cessation of respiration. Recovery.

(83.) Chloroform, of the strength 8 per cent., was given, by means of Mr. Clover's bag, to a rather small terrier dog. He became insensible at 1 min. 30 sec. At 3 min. 50 sec. the heart's action (indicated by the needle) had become irregular and intermitting. At 4 min. 40 sec. respiration ceased. At 5 min. 40 sec. galvanism was applied—the one pole being connected with the needle in the heart, the other with the prepuce. At this time the needle indicated regular, but feeble, pulsations. At 8 min. there was a slight effort at respiration, as pulse was felt in the femoral artery, and the heart evidently acted more strongly. The galvanism, however, was continued till 10 min., when the respiration was regular, but feeble, and the heart's action was strong. The animal recovered. In this case there was some little doubt whether effective galvanism was applied quite as soon as noted, as the apparatus had got out of order at the time.

Galvanism, after cessation of respiration. Unsuccessful.

(86.) Chloroform ($7\frac{1}{2}$ per cent.) was administered to a rather large dog, by means of Mr. Clover's bag. He was

insensible at 1 min. 15 sec. A few minutes later the respiration became feeble and irregular, and nearly ceased. At 7 min. 30 sec., however, he commenced to breathe freely. After this the respiration again failed, and finally ceased at 10 min. 35 sec. At 11 min. 35 sec. galvanism was applied; at this time the heart was still beating, but feebly, and there was a pulse in the femoral artery. The galvanism was applied over the two phrenic nerves in the neck. In the course of a few seconds the respiration again commenced. At 12 min. 25 sec., however, the needle in the heart ceased moving. At 13 min. 30 sec. slight movement of the needle in the heart was again observed; the respiration, however, was extremely feeble, and ceased a few seconds later. The galvanism was discontinued at 17 min. 30 sec., there being no evidence of life.

Artificial Respiration with Oxygen Gas. Recoveries.

(89.) Chloroform was given, by means of a towel, to a moderate-sized dog, and when insensibility was produced a tube was inserted into and tied in the trachea. He was allowed partly to recover, and at 13 min. the tube was connected with chloroform vapour. At 14 min. 40 sec. the respiration stopped; the heart at this time was beating slowly and imperfectly. At the time the artificial respiration was commenced (15 min. 40 sec.) the movement of the heart had almost, but not quite, ceased. At 15 min. 40 sec. artificial respiration was effected by means of a bladder of oxygen gas connected with the tube in the trachea. At 16 min. 40 sec. the pulsations of the heart had become much stronger. At 17 min. 45 sec. voluntary efforts at swallowing were made. At 18 min. 30 sec. the animal breathed freely, and sensibility returned.

(90.) Chloroform having been given to a rather small dog, a tube was inserted into and tied in the trachea. The animal was then allowed nearly to recover. At 6 min. 30 sec. chloroform was given, the trachea-tube being con-

nected with the vapour in a chloroform-bottle. At 7 min. 45 sec. the respiration, having ceased for 30 sec., again commenced, but at 12 min. 30 sec. it finally ceased, the heart still beating. At 14 min., the heart still beating steadily, artificial respiration with oxygen gas was commenced. This was accomplished by connecting the trachea-tube with a bladder containing the oxygen gas. At 17 min. natural breathing was established, and the animal soon after recovered. At 18 min. 40 sec. chloroform was again given, as before. At 20 min. 30 sec. the heart's action was very feeble, and the respiration slight. At 21 min. 20 sec. the respiration ceased for some time. The heart's action continued steadily. There were, however, no efforts at respiration till 27 min.; and at this time some feeble respiratory movements were observed. (It is possible that, in this experiment, some oxygen may have diffused itself into the lungs, as, after the cessation of the respiration, the trachea-tube was disconnected from the chloroform, and connected with the bladder of oxygen gas, so as to facilitate the commencement of artificial respiration.) The movements, however, were extremely slight, and only lasted a few seconds. At 28 min. 30 sec. all movement of the heart ceased. At 28 min. 40 sec. artificial respiration with oxygen gas was commenced. At 32 min. slight quivering movements of the tongue were observed, and at 34 min. there were efforts at respiration, the heart again beating. The animal recovered.

APPENDIX D.—STATISTICS

TABLE *of Amputations*

IN LONDON.

	Disease.			Accident.			Totals.		
	Cases	Died.	Per cent.	Cases	Died.	Per. cent.	Cases	Died.	Per cent.
University College, 1835 to 1846	133	24	...	64	27	...	197	51	...
St. Thomas's Hospital, 1835 to 1840	31	4	...	23	9	...	54	13	...
" 1842 to 1847	29	6	...	20	7	...	49	13	...
St. Bartholomew's, 1846	14	3	...	8	1	...	22	4	...
Guy's, 1843 to 1847	...	...	...	...	...	...	82	16	...
London Hospital, 1837 to 1842	109	36	...	77	33	...	186	69	...
Phillips, 1835 to 1838	...	...	...	...	...	...	233	53	...
Totals	316	73	23·1	192	77	40·1	823	219	26·6

OF SURGICAL OPERATIONS.

performed without Chloroform.

IN THE PROVINCES.

	Disease.			Accident.			Totals.		
	Cases	Died.	Per cent.	Cases	Died.	Per cent.	Cases	Died.	Per cent.
Liverpool Northern Hosp., 1834 to 1843	40	5	...	55	13	...	95	18	...
" 1845 to 1846	14	6	...	11	4	...	25	10	...
Glasgow Infirmary, 1794 to 1839	155	35	...	121	65	...	276	100	...
" 1839 to 1848	59	23	...	225	99	...	284	122	...
Newcastle Infirmary, 1823 to 1843	144	28	...	81	26	...	225	54	...
Chester Infirmary, 1838 to 1841	11	2	...	10	7	...	21	9	...
Exeter Hospital, 1816 to 1849	206	18	...	94	25	...	300	43	...
Radcliffe Infirmary, 1838 to 1847	69	12	...	22	2	...	91	14	...
Edinburgh Infirmary, 1840 to 1845	49	16	...	17	15	...	66	31	...
" 1846 to 1847	...	...	...	...	...	...	14	2	...
Aberdeen Infirmary, 1841 to 1846	27	6	...	15	5	...	42	11	...
Bristol, St. Peter's, 1844 to 1846	5	1	...	...	...	...	5	1	...
" Infirmary, 1845 to 1846	30	3	...	6	...	...	36	3	...
Derbyshire Infirmary, 1845 to 1846	11	2	...	...	...	...	11	2	...
Dundee Infirmary, 1844 to 1846	6	...	...	5	1	...	11	1	...
Elgin Infirmary, 1844 to 1846	9	2	...	1	1	...	10	3	...
Leicester Infirmary, 1845 to 1846	9	1	...	6	...	...	15	1	...
Perth Hospital, 1840 to 1847	22	8	...	...	...	...	22	8	...
Sussex County, 1844 to 1846	6	2	...	7	4	...	13	6	...
Salisbury, Stockport, Worcester, York, 1845 to 1846	25	2	...	21	8	...	46	10	...
Liverpool Infirmary, 1834 to 1836	...	...	...	...	...	...	43	3	...
Edinburgh Infirmary, 1839 to 1841	...	...	...	...	...	...	61	31	...
Six Scotch Hospitals, 1842	...	...	...	...	...	...	24	3	...
Reading Infirmary, 1838 to 1845	...	...	...	...	...	...	27	5	...
Provincial Totals	897	172	19·11	697	275	39·4	1763	491	27·8
London "	316	73	23·1	192	77	40·1	823	219	26·6
Grand Totals	1213	245	20·1	889	352	39·5	2586	710	27·4

TABLE *of Amputations*

IN LONDON.

	Disease.			Accident.			Totals.		
	Cases	Died.	Per cent.	Cases	Died.	Per cent.	Cases	Died.	Per cent.
St. George's, 1852 to 1859	115	29	...	34	12	...	149	41	...
St. Thomas's, 1857 to 1860	32	7	...	33	15	...	65	22	...
Guy's Hospital, 1853	...	...	...	...	...	...	25	2	...
Ditto, 1854 to 1861	148	21	...	96	46	...	244	67	...
Eight London Hospitals—Simpson	65	10	...	15	6	...	80	16	...
St. Bartholomew's, Jan., 1853, to Oct., 1863	228	46	...	130	28	...	358	74	...
Totals	588	113	19·2	308	107	34·7	921	222	24·1

TABLE *of Amputations performed with Chloroform.*

MILITARY.	Primary.			Secondary.			Totals.		
	Cases	Died.	Per cent.	Cases	Died.	Per cent.	Cases	Died.	Per cent.
Crimean War, to Nov., 1854	152	17	...	64	41	...	216	58	...
Ditto, from April, 1855	440	163	...	60	36	...	500	199	...
British Naval Brigade	45	14	...	18	9	...	63	23	...
	637	194	30·4	142	86	60·5	779	280	35·9
AMERICA.	Disease.			Accident.			Totals.		
Massachusetts Hospital	26	3	...	26	10	...	52	13	25·

performed with Chloroform—

IN THE PROVINCES.

	Disease.			Accident.			Totals.		
	Cases	Died.	Per cent.	Cases	Died.	Per cent.	Cases	Died.	Per cent.
Radcliffe Infirmary, from 1847	45	12	...	28	7	...	73	19	...
Leeds Infirmary, 1853 to 1861	110	17	...	79	28	...	189	45	...
Edinburgh Infirmary, 1850	19	5	...	8	3	...	27	8	...
Birmingham Hospital, 1859 to 1860	30	4	...	14	6	...	44	10	...
Glasgow Infirmary, 1849 to 1857	152	64	...	93	33	...	245	97	...
Newcastle Infirmary, 1846 to 1857	61	8	...	88	28	...	149	36	...
Twenty-four Provincial Hospitals	90	21	...	27	7	...	117	28	...
Fifteen ditto	59	10	...	23	8	...	82	18	...
Totals in Provinces	566	141	24·9	360	120	33·3	926	261	28·1
Totals in London	588	113	19·2	308	107	34·7	921	222	24·1
Grand Totals	1154	254	22·	668	227	33·9	1847	483	26·1

TABLE *of Amputations performed without Chloroform.*

MILITARY.	Primary.			Secondary.			Totals.		
	Cases	Died.	Per cent.	Cases	Died.	Per cent.	Cases	Died.	Per cent.
Fenwick—Guthrie	...	...	...	...	...	...	...	...	...
Alcock	...	...	...	...	...	...	1452	448	30·8
CONTINENT AND AMERICA.	Disease.			Accident.			Totals.		
Massachusetts Hospital	59	7	...	30	12	...	89	19	...
Pennsylvania Hospital	45	6	...	184	46	...	229	52	...
Phillips, 1834 to 1838	...	...	...	...	...	...	95	24	...
							413	95	23·
Hôtel Dieu, 1836 to 1842	...	...	...	...	...	...	178	104	...
Malgaigne	355	154	...	164	107	...	519	261	...
Phillips—France	...	...	...	...	...	...	203	47	...
„ Germany	...	...	...	...	...	...	109	26	...
							1009	458	45·3

Appendix E.

OBSTETRICAL REPORT.

The Effects of Chloroform in Obstetrical Practice, and in the Diseases of Women and Children.

The committee, in investigating the effects of chloroform in obstetric medicine, including the diseases of women and children, having determined that a series of direct experiments on the human subject would be surrounded by difficulties, considered that they would best promote the practical objects of their inquiry by collecting the accumulated experience of the profession on the various obstetrical uses of anæsthetics since their introduction.

A series of questions was therefore prepared and forwarded to all the teachers of midwifery and to the obstetrical officers of public institutions in the United Kingdom, requesting a record of their experience and opinions on the various matters indicated.

The following is an analysis of the replies:

A.—IN NATURAL LABOUR.

Question 1.—*Have you observed any instance of the occurrence of sudden death during the administration of chloroform in labour, or of the occurrence of symptoms indicating immediate danger to life?*

To this question 29 answers were received, all being in the negative, both in regard to the occurrence of sudden death or of symptoms indicating immediate danger to life. But in certain instances this negative was qualified by the observation that in certain cases some unfavorable symptoms had occurred, which, although not strictly dan-

gerous to life, may be briefly indicated as accidental to the employment of chloroform. Thus, one writer remarks that he had seen no death, but great exhaustion; another had observed in two cases rigidity of the lower limbs, with a sort of convulsive movement of the upper and of the face, which made him fear convulsions, but all ceased on suspending the inhalation. A third had met with a few cases of induced intermission of the pulse even under small doses of the vapour, and this had led him to prescribe a stimulant when the pulse is feeble. A fourth states that he had seen alarming faintness and swooning after delivery, without any hæmorrhage. A fifth remarks that in a few cases he had seen great prostration; and a sixth, that in a highly nervous woman the anæsthetic caused slight convulsions of an hysterical nature, and in another, who was very plethoric, a degree of coma which required active depletion.

Question 2.—When chloroform is given so as to produce deep anæsthesia, does it, according to your experience, interfere either with the uterine contractions or with the auxiliary powers of parturition?

An analysis of 28 answers to this question gives the following results:

In 8 it is stated to interfere with or lessen the uterine contractions.
In 3 „ „ „ the auxiliary powers.
In 14 „ „ „ both; whilst
In 3 it is stated not to interfere with either.

Question 3.—When administered in a moderate degree and under proper regulation, has it, in your practice, protracted labour by weakening the expulsive powers, either in the early or later stages of parturition?

Twenty-seven answers were received to this inquiry, to the following effect:

In 4 it is said to weaken them in the early stage.
In 2 „ „ „ later stage.
In 12 „ „ „ in both; and
In 9 „ not to weaken them in either.

Question 4.—Has it any beneficial effect in promoting the dilatation of the maternal passages?

Of 26 answers to this inquiry, 22 were in the affirmative, and 4 only in the negative.

Question 5.—Has its employment during natural labour predisposed to puerperal convulsions, apoplexy, or other complications on the part of the mother?

Twenty-seven answers were received in reply to this question, and, with 3 exceptions, in the negative. Of the 3 exceptional replies, one writer refers to 2 cases in which slight convulsive movements occurred during its employment, and ceased on its withdrawal. Another had observed hæmorrhage to occur when long given, and apparently from its relaxing effect upon the uterus; and a third had observed dangerous fainting to occur after delivery without hæmorrhage. On the other hand, two writers speak of it as having the power of lessening the tendency to convulsions; and one of them states that, in his experience, it has warded them off in several cases.

Question 6.—Do you believe that the number of cases in which instruments must ultimately be used to terminate labour is likely to be increased from the employment of chloroform?

Fifteen answers were returned to this question in the negative, and 9 in the affirmative. But, as regards the latter, it should be stated that this result was only apprehended by some of the respondents when the drug was given largely, just as, in regard to the former, it was assumed that the drug had been given cautiously and judiciously.

Question 7.—Has it, in your experience, predisposed to imperfect contraction of the uterus after delivery, and thus led to post-partum and secondary hæmorrhage?

To this question 29 answers were returned. One gentleman expresses himself doubtfully in the negative; 13 aver that it predisposes to both, and 15 that it predisposes to neither.

Question 8.—*Has it had any such after-effects on the nervous or vascular system of the mother as to retard her convalescence, or render her more liable to any of the forms of puerperal disease*—e. g. *puerperal fever, phlegmasia dolens, puerperal mania?*

Of 29 answers received to this question, 26 were distinctly in the negative, and in several it was stated to have a directly contrary effect to that of retarding convalescence. Thus, one writer remarks that it appears to him to have a "precisely contrary effect;" another has found convalescence to have been more favorable; a third remarks that the convalescence has been manifestly better; a fourth, that it promotes convalescence; a fifth says that, so far from retarding convalescence, in his experience it has been the reverse; and a sixth, that it aids and expedites recovery.

On the other hand, three answers were returned in the affirmative; in one, it is stated that a lady was amaurotic for several hours after labour; in another that two or more attacks of puerperal mania had followed its use; and in a third that convalescence had been retarded, apparently from its effect on the nervous system.[1]

Question 9.—*Has it had any tendency, from its after-effects, to interfere injuriously with the function of lactation?*

In reply to this query, 27 answers were received in the negative, and one in the affirmative.

[1] No reference is made in any of the replies to the subsequent occurrence of sickness as a drawback to the employment of chloroform in obstetrical as distinguished from surgical practice.

Question 10.—*Has it any injurious influence on the child*

Of 29 answers received, 27 were in the negative. In one it was stated that the writer was not in possession of sufficient facts to answer the question, and in one that the respondent had observed great drowsiness and a disinclination on the part of the child to take the breast for two or three days.

B.—IN ABNORMAL LABOUR.

Question 1.—*Have you employed chloroform in any obstetrical operations? If so, what advantages do you think you have gained by its employment, and what evils have seemed to you to detract from its value?*

(e. g.) *In cases where the patient is much enfeebled by hæmorrhage, and turning or instruments must be employed, is the use of chloroform, in your opinion, to be regarded as the introduction of a further element of danger, and likely to diminish the chances of ultimate recovery?*

An analysis of 29 replies to this question gives the following result:—25 of the respondents had employed it with advantage, 2 had not used it, and 2 had employed it and found its action unfavorable. Amongst the cases instanced in which it had proved beneficial are—turning; the application of the forceps, especially in a narrow pelvis; the extraction of retained placenta, and craniotomy: and the advantages claimed for it are—that the patient is passive instead of resisting under treatment; that the operator is not disturbed by her outcry; that the shock is reduced to a minimum; that it lessens resistance by relaxing rigid tissues, and that it favours convalescence by preventing pain, and consequently exhaustion. On the other hand, in the two instances in which it was reported upon unfavorably, it was stated in one that the writer had found it disadvantageous

in forceps cases, from the turning over of the patient; and, on the other, that in forceps and craniotomy cases the woman had been always more unmanageable because unconscious. As regards the propriety of using it in these cases, when the patient is much enfeebled by hæmorrhage or other causes, 17 answers were received unfavorable to its employment, whilst in 4 a contrary opinion was expressed, one of the latter being qualified by the remark "provided opium and stimulants were given."

Question 2.—Have you employed chloroform in the treatment of puerperal convulsions? If so, do you believe that it may enable the practitioner sometimes to dispense with other aids (such as bleeding, &c.) occasionally used in such cases?

Twenty-eight replies were received to this question, which may be thus tabulated:—14 of the respondents had used it and found it beneficial, 11 had not tried it, 1 had tried it and found it not beneficial, and 2 returned qualified answers in its favour. Of the 13 affirmative replies, the following may be regarded as an embodiment of the principal information furnished. One writer observes that, except in slight cases, he had never ventured to dispense with other treatment, but that he had repeatedly seen it arrest the convulsion both in forceps and craniotomy cases. Another had used it beneficially, but in most cases after bleeding; when bleeding, however, is improper, it quiets the patient, although the writer would not trust to it exclusively. A third had used it with the best effects without having used the lancet. A fourth had used it beneficially in one case, in which, from the weakness of the patient, he was opposed to bleeding. A fifth had used it with advantage in forceps cases, but thinks that care should be taken in giving it when much insensibility is present. A sixth had found it useful, but not to the exclusion of other aids, and more especially with the view of arresting post-partum convulsions until sleep had taken place. A seventh states that he had used it with

signal advantage in several cases, and had found it to supersede bleeding entirely; and he further adds, that it acts as a preventive in patients who had previously had them. An eighth had repeatedly given it with striking success, but doubts if it supersedes the use of bleeding where the pulsation of the temporal arteries is firm and strong, and especially if the patient sees sparks or flashes of light before her eyes. A ninth had used it with great success; but thinks that bleeding, and other means with it, might be both prudent and proper. Of the two qualified answers received, in one the writer observes that he thinks he has sometimes seen it useful, but is sure that it acts injuriously when respiration is imperfect, the face blue, and the breathing short and interrupted; and in the other it is stated that everything must depend upon the type of the convulsions, and that usually he would not give it before resorting to bleeding and other depletory means. On the other hand, one writer states that he had only used it once, that it did no good, and that he would not trust to it without other means.

C.—MODE OF ADMINISTRATION; QUESTION AS TO THE USE OF ETHER.

Question 1.—*Have you any reason for giving a preference to ether over chloroform in obstetrical practice?*

Sixteen answers were received to this question, and all in favour of chloroform; but in two instances a qualified preference was given to ether where chloroform disagreed. Thus, one writer remarks that he gives a preference to ether when chloroform disagrees by producing sickness, &c., or when it is especially disliked by the patient, and another expresses himself to the same effect. On the other hand, three of the answers were decidedly in favour of chloroform, one writer preferring it on account of its more certain effect; another stating that he had twice given ether, but with disappointment, and remarking that it was much less efficacious;

whilst a third affirms, that he would not under any circumstances use ether, inasmuch as he had tried it in a few cases, and had found it to be a most disagreeable and dangerous agent.

Question 2.—What rules have you observed, or would you be disposed to recommend, for the use of anæsthetics in natural and morbid labour?

The general bearing of the answers to this question is to the effect that, as regards natural labour, chloroform should be given sparingly, and so as to alleviate rather than to extinguish the pains. With this view a great many of the respondents recommend that it should only be given at the commencement of each pain; others, that it should only be given if the pains are very severe; and others, that it should only be given in the latter stages of labour. On the other hand, it is generally recommended that in morbid, and especially in instrumental or operative, labour, it should be given freely. The rules for its safe administration may be stated as deduced from different answers:—1. Avoid giving it directly after a meal. 2. In primiparæ especially give it very moderately. 3. Dilute well with air and watch the pulse and the breathing. 4. Keep your finger constantly on the pulse, and the moment it fails, discontinue the chloroform. 5. Give it slowly. 6. If depressed, give an occasional stimulant. 7. When the head bears upon the perinæum, give it more freely, to promote relaxation. 8. In excitable persons, unless it acts well, it is better not to use it. 9. Always suspend its administration towards the end of labour. 10. Do not continue its use for a prolonged period if not absolutely necessary. 11. When deep anæsthesia is required it is best to have a skilled administrator. 12. With this object give it slowly, and if it causes delirious excitement withhold it. 13. In ordinary cases administer only as much as will render the patient indifferent to pain rather than unconscious of it, and give a little brandy at intervals.

Question 3.—*Have you any reasons for believing that a special apparatus is desirable in obstetric practice? What, in your experience, has appeared to be the best method of exhibition?*

To the first part of this inquiry 22 answers were received in the negative, and 4 in the affirmative. Of the latter one respondent states that he prefers an apparatus which gives some degree of certainty as to the per-centage in combination with air. A second remarks that, although he does not consider a special apparatus necessary, yet he thinks that a Snow-form of apparatus is desirable. A third uses and recommends an apparatus designed by himself, and a fourth thinks it is best to have a special apparatus when the patient is to be placed fully under its influence.

Of the 22 negative replies, the majority of the writers use and recommend merely a pocket-handkerchief or towel, some place pieces of lint or sponge moistened with chloroform into a tumbler or shallow cup, and one or two recommend an adjustment of the handkerchief or towel in a peculiar way. It should be added that some of these writers deprecate very strongly the use of a special apparatus.

D.—DISEASES OF WOMEN AND CHILDREN.

Question 1.—*The use of chloroform in the diagnosis of diseases of women* (*spurious pregnancy, hysteric tympanitis, and other allied affections*).

Fifteen answers only were received to this question, in one of which the writer states that he had not used it in these cases, whilst in the other 14 it is stated to be highly useful. The cases in which its utility is represented to be greatest are chiefly—the diagnosis of spurious pregnancy and phantom tumours; the facilitating of examination of the uterine organs where there is much sensibility or intolerance of pain;

cases of feigned disease; the diagnosis of abdominal or pelvic tumours; certain forms of dysmenorrhœa; and in some hysterical affections.

Question 2.—The use of chloroform in the treatment of spasmodic diseases of women and children.

Fourteen replies of a favorable character were received to this question, and on the other papers either no answer is given or it is stated that the anæsthetic had not been tried. Of the 14 answers which were in favour of its efficacy, the following are some of the cases in which its utility had been favorably tested:—Hooping-cough, especially when complicated with convulsions; some laryngeal affections, when there is much spasm of the glottis; spasmodic croup; epileptic seizures; hysterical convulsions in women; some forms of convulsion in children arising from no other cause than cerebral irritation; hysterical local muscular contractions, and wry-neck.

The thanks of the committee are due to the Council of University College for their liberality and courtesy in allowing the experiments to be made in the physiological laboratory of that institution. They also wish to express their cordial thanks to Mr. Clover, who, although not a member of the committee, attended, at their request, nearly all the meetings for experiments, administered the chloroform, and contrived, from time to time, with remarkable ingenuity, special apparatus for carrying them on.

The committee desire to record their thanks to those members of the profession who have contributed important communications respecting accidents with chloroform, and observations concerning its administration in surgical and obstetric practice; also to those surgeons of the hospitals

and to the chloroformists by whom the various mixtures of chloroform and ether have been tested; to Drs. Parker, Richardson and Kidd, and to MM. Potter, Clover, and Bader, for the valuable information they have given respecting the practical administration of anæsthetics; and to Dr. Althaus, for kindly assisting at some of the experiments on resuscitation.

(Signed)—

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INDEX.

PAGE

Absorption of dead bone (Mr. Savory) . . . 103
Amputations performed at St. Bartholomew's Hospital, 1853-63 (Mr. Callender) 75
Aneurismal clamps, application of indices to (Mr. E. Hart) . 31
—— tumours, implication of branches of pneumogastric nerve in (Dr. Habershon) 35
Aneurisms, incurable, method of procuring the consolidation of fibrin in (Mr. Moore) 129
Aneurism of the ascending aorta treated by insertion of wire (Mr. Moore) 129
—— ditto, case of ditto (Dr. Murchison) . . . 136
—— of the abdominal aorta, cured by compression immediately above the tumour (Dr. W. Murray) 187
—— popliteal, unusual occurrences during the cure of (Mr. Moore) . 17
—— ditto, cured by flexion of the knee (Mr. Durham) . . 25
—— ditto (Mr. H. C. Johnson and Mr. Hart) . . . 29
Aorta, abdominal, aneurism of, cured by compression immediately above the tumour (Dr. W. Murray) . . . 187

Bickersteth (Mr. E. R.) new operation for obtaining union of ununited fracture, and its application to cases of recent fracture . 113
Bilharzia Capensis, description of the parasite . . . 59-63
Bladder, cancerous ulcer of the, in case of cancer of the penis . 1
Bone, absorption of dead (Mr. Savory) . . . 103
Brodie (Dr. G. B.) statistics of Queen Charlotte's Lying-in Hospital 169
Bryant (Mr. Thomas) two cases of stone in the bladder of the female treated by rapid urethral dilatation . . . 151
—— tables of cases 163-167

Calculus in the female bladder, treated by rapid urethral dilatation, &c. (Mr. T. Bryant) 151
—— tables of cases, expelled by natural efforts, removal by dilatation, removal after incising urethra, and removal by lithotrity 163-167

PAGE

CALLENDER (Mr. G. W.) amputations performed at St. Bartholomew's Hospital, 1853-63 75
Cancer, secondary deposits, in case of cancer of the penis . . 1
Cancerous infiltration of the penis, with secondary deposits in other parts (Mr. H. Coote) 1
—— ulcer of bladder, in case of cancer of the penis . . 1
Cape of Good Hope, endemic hæmaturia of (Dr. John Harley) . 55
CHLOROFORM, *Report* of the Committee on its uses, effects, and administration 323
—— *Appendix A*, selected experiments on the cessation of respiration and the heart's action, respiratory movements, &c. . . 360
———— *B*, accidents with chloroform (with Tables A to H) . . 370
———— *C*, selected experiments for resuscitation . . . 416
———— *D*, statistics of surgical operations . . . 429
———— *E*, Obstetrical Report 432
—— how it arrests animal life 324
—— effect of, on the heart 326
—— effect of, on respiration 329
—— effect of division of the pneumogastric nerves in animals under its influence, &c. 331
—— action of, on the glottis and fauces . . . 332
—— post-mortem appearances in animals poisoned with . . 336
—— means of avoiding accidents with . . . 339
—— mode of administration 343
—— rules relating to the administration of . . . 352
—— use of, in surgical operations 354
—— use of, in obstetric practice 356
—— and ether, effect of the mixture of . . . 339
Clamps, aneurismal, application of indices to (Mr. E. Hart) . 31
Compression, aneurism of the abdominal aorta cured by, above the tumour (Dr. W. Murray) 187
COOTE (Mr. Holmes) cancerous infiltration of the penis, with secondary deposits in other parts 1
Cyst, mucous, on the laryngeal aspect of the epiglottis, treated by incision (Mr. Durham) 7
Cystic tumours of the ovary, their origin, structure, and development (Dr. W. Fox) 227

Dunsterville (Mr. G.) particulars relative to the endemic hæmaturia of the Cape of Good Hope 64
DURHAM (Mr. Arthur E.) mucous cyst on the laryngeal aspect of the epiglottis, treated by incision 7
—— popliteal aneurism cured by flexion of the knee . . 25

PAGE
Epiglottis, mucous cyst on the laryngeal aspect of, treated by incision (Mr. Durham) 7
Erichsen (Mr. John) note on a case of unusual difficulty in lithotomy from distortion of the pelvis . . . 11
Ether, how it arrests animal life 333
—— effect of, on the heart 335
—— effect of, on respiration 335
Ether and chloroform, effect of the mixture of . . . 339

Fenwick (Dr. S.) on the condition of the stomach and intestines in scarlatina 209
Fibrin, new method of procuring the consolidation of, in incurable aneurisms (Mr. Moore) 129
Flexion of the knee in popliteal aneurism (Mr. Durham) . . 25
—— ditto (Mr. H. C. Johnson and Mr. Hart) . . . 29
Fox (Dr. Wilson) origin, structure, and mode of development of cystic tumours of the ovary 227
Fracture, ununited, new operation for obtaining union of (Mr. Bickersteth) 113

Greenhow (Dr. E. H.) congenital imperfection of the mammæ, sexual organs, sternum, and heart, in a woman aged twenty-two . 195

Habershon (Dr. S. O.) implication of branches of the pneumogastric nerve in aneurismal tumours 35
Hæmaturia, endemic, of the Cape of Good Hope (Dr. John Harley) . 55
—— notices of ditto by Mr. G. Dunsterville . . . 64
—— other notices by Dr. J. W. Johnston, Mr. J. Henderson, Mr. G. Saunders, &c. 69
Harley (Dr. John) endemic hæmaturia of the Cape of Good Hope . 55
Hart (Mr. E.) Mr. H. C. Johnson's case of popliteal aneurism cured by flexion of the knee 29
—— on the application of indices to aneurismal clamps . . 31
Heart, effect of chloroform on the 326
—— movements of, after the rhythmic contractions have ceased . 328
—— &c., congenital imperfection of 195
Hernia, on the causes of (Mr. Kingdon) . . . 295
—— strangulated femoral, neck torn from the body of the sac, and formation of sub-peritoneal pouch by escaping bowel (Mr. Hulke) 97
Hospital, St. Bartholomew's, amputations performed at, 1853-63 (Mr. Callender) 75

PAGE

HULKE (Mr. J. W.) strangulated femoral rupture, neck torn on a former occasion from the body of the sac, and formation of sub-peritoneal pouch by escaping bowel . . . 97

Imperfection, congenital, of mammæ, sexual organs, sternum, and heart in a woman aged twenty-two (Dr. Greenhow) . . 195
Indices, their application to aneurismal clamps (Mr. E. Hart) . 31
Intestines, condition of, in scarlatina (Dr. Fenwick) . . 209

JOHNSON (Mr. H. C.) case of popliteal aneurism cured by flexion of the knee 29

KINGDON (Mr. J. A.) on the causes of hernia . . . 295

Lithotomy, unusual difficulty in, from distortion of the pelvis by rickets (Mr. H. Thompson) 11
Lying-in hospital, statistics of Queen Charlotte's (Dr. Brodie) . 169

Mammæ, &c., congenital imperfection of, &c. (Dr. Greenhow) . 195
Meatus, external auditory, sebaceous tumours in (Mr. Toynbee) . 203
MOORE (Mr. C. H.) unusual occurrences during the cure of a popliteal aneurism 17
—— new method of procuring the consolidation of fibrin in incurable aneurisms, with case by Dr. Murchison . . . 129
MURCHISON (Dr. C.) case of aneurism of the ascending aorta treated by an insertion of wire (on Mr. Moore's method) . 129, 136
MURRAY (Dr. Wm.) case of aneurism of the abdominal aorta, cured by compression of that artery immediately above the tumour . 187

Nerve, pneumogastric, implication of branches of, in aneurismal tumours (Dr. Habershon) 35

Ovary, cystic tumours of, their origin, structure, and development (Dr. W. Fox) 227

Paracentesis capitis, successful case of (Mr. T. Y. Thompson) . 289
Parasite, Bilharzia Capensis, description of . . 59-63
Pelvis, great distortion of, and difficulty from, in case of lithotomy (Mr. H. Thompson) 11
Penis, cancerous infiltration of (Mr. H. Coote) 1
Pneumogastric nerve, implication of branches of, in aneurismal tumours (Dr. Habershon) 35

PAGE

REPORT of the Committee on the uses and physiological, therapeutical, and toxical effects of chloroform and its administration . 323
Resuscitation in cases of poisoning by chloroform inhalation . 344
Rupture, see *Hernia*.

St. Bartholomew's Hospital, amputations performed at, 1853-63 (Mr. Callender) 75
SAVORY (Mr. W. S.) on the absorption of dead bone . . 103
Scarlatina, condition of the stomach and intestines in (Dr. Fenwick) . 209
Sebaceous tumours in external auditory meatus (Mr. Toynbee) . 203
Sexual organs, &c., congenital imperfection of . . . 195
Statistics of Queen Charlotte's Lying-in Hospital (Dr. Brodie) . 169
Sternum, &c., congenital imperfection of . . . 195
Stomach and intestines, condition of, in scarlatina (Dr. Fenwick) . 209
Stone in the female bladder, treated by rapid urethral dilatation (Mr. T. Bryant) 151

THOMPSON (Mr. Henry) unusual difficulty in lithotomy from distortion of the pelvis by rickets 11
THOMPSON (Mr. T. Y.) successful case of paracentesis capitis . 289
TOYNBEE (Mr. Joseph) sebaceous tumours in external auditory meatus ('Suppl. to Seventh Series on Diseases of the Ear') . . 203
Tumours, cystic, of the ovary, their origin, structure, and development (Dr. W. Fox) 227
—— sebaceous, in external auditory meatus (Mr. Toynbee) . 203

Union, new operation for obtaining, in ununited fracture (Mr. Bickersteth) 113
Urethral dilatation, rapid, in stone in the female bladder (Mr. T. Bryant) 151

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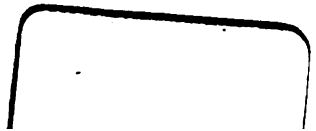
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